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THE ORIGIN AND DESTINATION OF CERTAIN AFFERENT AND EFFERENT TRACTS IN THE MEDULLA OBLONGATA.

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(1) INTRODUCTION.

THE experimental researches on which this paper is based were carried out in the Pathological Laboratory of University College, when I was Research Scholar to the British Medical Association, and during the time that the

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laboratory was under the directorship of Professor Victor Horsley; I therefore take this opportunity of thanking him for the privilege which I then enjoyed. The microscopical examination of the central nervous system of the animals then operated on has been made subsequently.

The question with which I specially intend to deal is that with regard to the existence of efferent tracts in the spinal cord other than those belonging to the pyramidal system.

In addition to this question of efferent tracts in the spinal cord, the subject of afferent tracts degenerating in the medulla, pons and mesencephalon, after various lesions of the medulla, will also be discussed, in view of the results which have been obtained in this connection.

I do not propose to refer in detail to the extensive literature relating to the questions about to be dealt with in this paper, but intend to allude to those investigations, more especially recent ones, which bear directly on the points which I have investigated. This will be done in the discussion which will be found in connection with each part of the paper after the results of the experiments have been detailed, and also in a more general discussion on the question of efferent tracts in the spinal cord. The results of Biedl[1] will, however, be rather more fully dealt with, in that this observer has called into question certain of my negative results in connection with ablation of the cerebellum,[2] which have a direct bearing on the questions about to be discussed in this paper.[3]

(2) Methods of Research.

Dogs and monkeys (Macacus rhesus and sinicus) were alone employed in the investigations which form the subject of the communication. In every instance the animal was

[1] Biedl, *Neurolog. Centralblatt*, 1895, vol. xlv., p. 434.

[2] Risien Russell, *Phil. Trans. Roy. Soc.*, 1894, vol. clxxxv., B., p. 819.

[3] Since this paper was written an important monograph by Dr. André Thomas has appeared, entitled "Le Cervelet, étude anatomique, clinique et physiologique," Paris, 1897. This observer confirms Marchi's results as regards the existence of an efferent antero-lateral tract in the spinal cord, derived directly from the cerebellum.

rendered unconscious by the administration of ether by inhalation, and narcosis was maintained throughout the operation. The hair was shaved from the back of the head and neck, the skin then cleansed by soap and water, and subsequently thoroughly disinfected by perchloride of mercury lotion (1 to 1,000). A median incision was made from the superior occipital protuberance to about the mid-cervical region posteriorly, and the muscles were then scraped from their attachments to the occipital bone and to the upper cervical spines and arches. Bone forceps were then used to expose the posterior portion of the cerebellum, the medulla and upper part of the cervical cord, up to which point instruments first sterilised by boiling, and subsequently kept in carbolic lotion (1 to 40), were used, and the wound was washed with perchloride of mercury lotion (1 to 2,000), sterilised cotton wool being used instead of sponges. As soon as all bleeding had been arrested and the dura mater was about to be opened, boiled normal saline solution was used to freely wash out the wound, and from this point in the procedure no antiseptic lotion was employed, the instruments and the cotton wool used for sponges being both kept in boiled normal saline solution. On the importance of this I have sufficiently insisted elsewhere[1] which makes it unnecessary for me to do so again here. The posterior part of the middle and one lateral lobe (in every instance the left) of the cerebellum were gently raised by means of a small elevator, and if my object was to divide the inferior peduncle of the cerebellum as high up as possible, or to cut out Deiters' nucleus, then the cerebellum had to be raised considerably more than in those instances in which a lesion to the lateral region of the medulla, or to the posterior columns and their nuclei, was to be produced. In the case of the lateral medullary lesion it was necessary to gently displace the medulla laterally towards the opposite side, and to rotate or raise it slightly to enable me to produce the lesion sufficiently ventrally to avoid injury to the restiform body. Every care was, of course, taken not to injure the spinal accessory or hypoglossal roots. A delicate,

[1] *Loc. cit.*

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narrow-bladed knife was used to divide the peduncle, to cut out Deiters' nucleus, and to divide the posterior columns and their nuclei; this was also used to divide the lateral tracts of the medulla in some instances, but on other occasions the lesion of the medulla was produced by inserting a needle just dorsally to the position of the inferior olive, pushing it about two to three millimetres towards the cephalic end of the medulla and then moving the point of the needle so inserted from side to side in different directions so as to destroy the region into which the needle point had entered, every care being, of course, taken to prevent injury to the pyramids, both when the knife was used and when the needle was substituted.

The skin wound was subsequently sutured by means of sterilised horse-hair, and an antiseptic dressing applied after further washing of the skin surface with an antiseptic lotion. Three weeks to a month after this the animals were killed by an overdose of chloroform, administered by inhalation, the central nervous system at once removed and placed in Müller's fluid, and subsequently prepared for microscopical examination by means of the Marchi method, which is now too well known to call for special description here.

(3) Plan of Research.

The following experimental procedures were adopted in attempting to arrive at the facts with regard to the origin and destination of some of the afferent and efferent tracts in the medulla oblongata.

(1) Section or destruction of the lateral region of the medulla oblongata between the ascending root of the fifth nerve and the inferior olive.

(2) Division of the restiform body.

(3) Division of the direct sensory cerebellar tract of Edinger.

(4) Severance of Deiters' nucleus from its connection with the medulla.

(5) Section of the posterior columns and their nuclei in the medulla.

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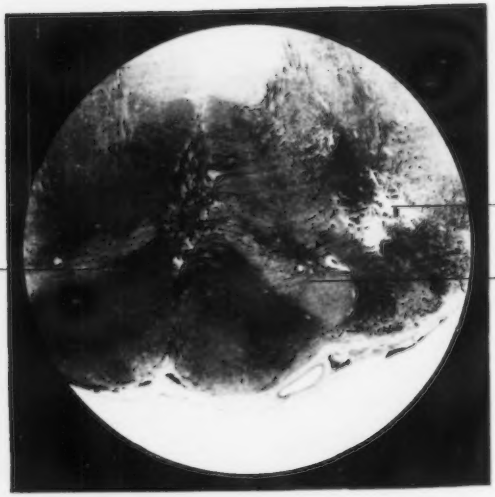

FIG. 1.

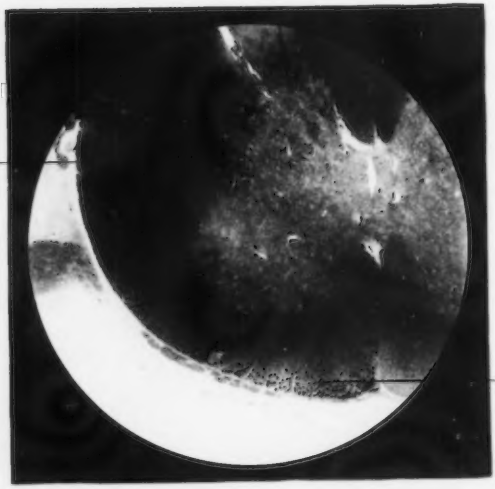

FIG. 2.

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Part I.—Section or Destruction of the lateral region of the Medulla Oblongata between the Ascending Root of the Fifth Nerve and the Inferior Olive.

As may be seen by reference to fig. 1, the lesion produced in the lateral region of the medulla is so situated that the inferior olive has just escaped injury and there is no sign of any injury to either pyramid; indeed, the way in which I produced the medullary lesion precluded any possibility of injury to the pyramid without first injuring the olive. In the section from which the photo-micrograph has been taken the only degeneration present, other than that in the direct neighbourhood of the lesion, is a well marked degeneration of the opposite fillet seen lying immediately dorsal to the opposite pyramid, a degeneration obviously due to interruption of the arciform fibres, by the medullary lesion, in their passage from the posterior column nuclei on the side of the lesion to the opposite fillet. As will subsequently be seen, the absence of injury to the opposite pyramid is of even more importance in relation to the subject under discussion than is the fact that the pyramid on the side of the lesion is intact.

(*a*) *Efferent Tracts.*—From the lesion in the lateral region of the medulla a well marked descending degeneration may be traced into the spinal cord; that this extensive band of degeneration consists of two distinct efferent tracts will subsequently become evident, but in the cervical region of the spinal cord from which the photo-micrograph represented in fig. 2 was taken, the degenerated fibres apparently constitute a single extensive tract, which, commencing dorsally in a well marked triangular area of degeneration, situated just ventrally to the crossed pyramidal tract and internally to the anterior part of the direct ascending cerebellar tract and the posterior part of the afferent antero-lateral tract of Gowers, sweeps round the periphery of the antero-lateral region of the cord to the anterior median fissure. Continuous as this area of degeneration appears to be, there are two regions in which it is more extensive than elsewhere; one of these is its posterior limit, where it is

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triangular in shape, the apex of the triangle pointing inwards towards the gray matter of the lateral horn, and ventrally at a point just external to the anterior fissure where the band of degeneration becomes nearly twice the depth of the narrow band situated at the periphery of the cord and connecting these two points of more extensive degeneration. As may be seen in fig. 2, the degeneration does not extend to any extent into the region of the direct pyramidal tract as it abuts on the anterior median fissure, or indeed into any part of the area of the cord occupied by the direct pyramidal tract in man, as I hope to be able to show by comparison subsequently. A cursory examination of fig. 2 suffices to convince one that the crossed pyramidal tract is encroached on by the degenerated fibres forming the posterior limit of the triangular area of degeneration already alluded to.

As the thoracic region of the spinal cord is reached it becomes more and more evident that the area of degeneration under consideration is really composed of two tracts; the narrow band of degenerated fibres which connects the posterior triangular area with the area situated just external to the anterior tip of the anterior median fissure, becomes gradually thinned out (see fig. 3) until a breach of continuity becomes evident, separating the area of degeneration situated ventrally from that situated dorsally to it. The dorsal area of degeneration, while still retaining some evidence of the triangular form so well seen in the cervical region of the cord, now becomes much more scattered and thinned out, a few degenerated fibres are scattered towards the region of the crossed pyramidal tract, but by far the greater tendency of spread is in the shape of a narrow band occupying the periphery of the cord external to this tract. Very little alteration is to be noted in the area of degeneration close to the anterior tip of the anterior median fissure, which occupies an area of almost the same breadth as in sections taken from higher levels, and shows no more evidence of extending along the margin of the anterior fissure or in any other way encroaching on the domains of the direct pyramidal tract. Evident as was the separation

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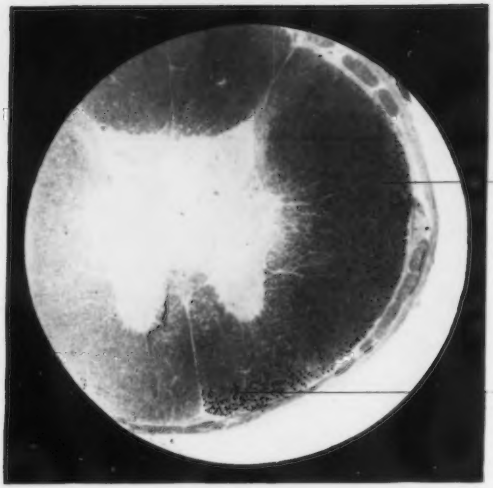

FIG. 3.

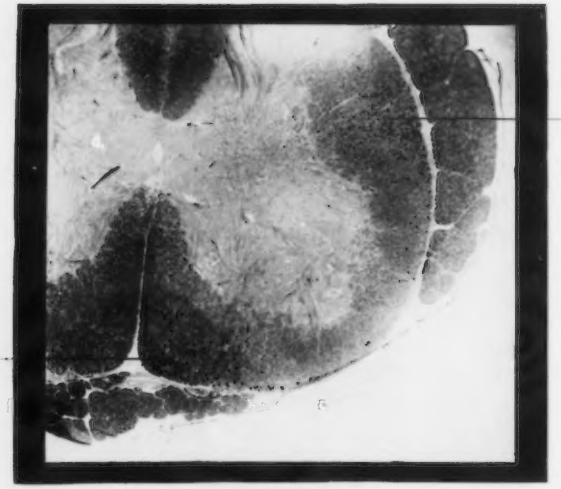

FIG. 4.

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between the two tracts on examination of the thoracic cord, the picture presented by sections taken from the lumbar region of the cord, and shown in fig. 4, leaves no possible room for doubting that we are dealing with two separate and distinct tracts of degenerated fibres. The whole of the narrow band of degeneration occupying the periphery of the cord, so well seen in the cervical region connecting the two major areas of degeneration, has now completely disappeared and there remain two distinct and separate tracts, the one situated in close proximity to the crossed pyramidal tract, and the other at the periphery of the cord close to the anterior median fissure. The posterior area now forms a much more compact bundle than was the case in the thoracic region, and it again assumes most distinctly the triangular form so characteristically seen in the cervical region of the cord; but instead of the base of this triangular area of degeneration being separated from the periphery of the cord by a distinct band of undegenerated nerve fibres, as was the case in the cervical region, it now comes quite to the surface in the greater part of its extent, only a limited portion of the tract being separated from the margin of the cord posteriorly by undegenerated fibres. The apex of the triangular area, as before, points inwards towards the grey matter of the cord. So nearly does this area of degeneration in the lumbar cord correspond to the region occupied by the crossed pyramidal tract, that there must be considerable intermingling of the two systems of fibres.

As regards the ventral tract situated close to the anterior median fissure, there has been a marked thinning out of its fibres, and while the chief seat of degeneration is that which it occupied at higher levels, there is, nevertheless, a little more tendency for the degenerated fibres to spread along the margin of the anterior fissure, but they only extend to rather less than half way to the anterior commissure, and even up to that point are by no means plentiful.

Before considering this anterior tract further it will be convenient to dispense with the lateral tract that has occupied our attention. Throughout its course in the spinal cord degenerated fibres may be traced from it to the

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grey matter of the anterior horn, the bulk of these fibres apparently passing to the part of it known as the lateral horn. No evidence has been obtained that these fibres decussate at any point in their course from the lesion in the medulla; they maintain a direct course on the side of the lesion throughout the spinal cord, and only pass to the grey matter of that side of the cord; none of the fibres could be traced across to the opposite side even in the grey matter.

That the tract under consideration is probably identical with that which was described by Boyce[1] in the cat after hemisection of the mesencephalon, in which his lesion passed between the anterior and posterior quadrigeminal bodies, is rendered extremely probable by a comparison of fig. 2 with fig. 5, plate 3, in Boyce's monograph on the subject. The position, shape, and general characters of the tract, as seen in the cervical region, are practically identical with that described by Boyce; there is, however, this difference with regard to our observations, Boyce could find no evidence of the existence of his tract caudal to the upper thoracic region of the spinal cord where it appeared to end, whereas, as has been seen, the tract to which I am now calling attention has been traced throughout the spinal cord to the lowest part of the lumbar region. This may indicate a fundamental difference between the two tracts, as is further suggested by the fact that the tract described by Boyce is one originally derived from a decussating system, whereas at no point in its course from the medulla throughout the spinal cord have I been able to find any evidence that the tract I am describing decussates. In spite of these apparent differences I am inclined to regard the tract I have found as belonging, at any rate, to the same system of fibres as that described by Boyce, even if it cannot strictly be regarded as the continuation and termination of that tract.

A similar tract has been described by Ferrier and Turner[2] as degenerating from the nucleus lemnisci lateralis, leaving the lateral fillet at the level of the motor nucleus

[1] *Phil. Trans. Roy. Soc.*, 1895, vol. clxxxvi., B., p. 321.

[2] Ferrier & Turner, *Phil. Trans. Roy. Soc.*, 1894, vol. clxxxv., B., p. 719.

of the fifth nerve, and traced by them as far as the sixth thoracic segment of the spinal cord.

Boyce found that the degenerated fibres which formed the tract in question passed from the side of the lesion to the opposite side in Forel's decussation and then passed caudalwards, and Held[1] speaks of fibres originating in the red nucleus, which pass caudalwards to form the lateral column. It is quite conceivable that an "internuncial" system, even of long fibres, may only extend for a limited distance of the full length of that system, and that there should be a constant accession of fibres to the system originating at levels further caudal to the level at which the highest fibres arise, and extending further caudalwards than these fibres, though not of necessity longer than the fibres arising farthest cephalwards and which belong to the same system.

That this is not too great an assumption is made evident by the fact that even in the pyramidal system such an accession occurs; thus in a cat whose nervous system I examined microscopically[2] and in whom the pyramid was completely absent on one side, as seen in sections through the mesencephalon, pons and upper part of the medulla, fibres entered this system from lower levels, and were not derived by decussation from the opposite intact pyramid. The evidence obtained in this animal was to the effect that there is an accession of fibres to the pyramidal system as lower and lower levels are reached, and that all the fibres contained in the pyramids are not derived from the cerebral hemispheres. The same condition has been found to occur in man.

If such a state of things is possible in connection with a system like that of the pyramidal fibres, how much the more likely is it to occur in connection with an "internuncial" system, such as that which we have been considering.

That a system of fibres other than pyramidal ones existed in close relationship to the crossed pyramidal tracts was

[1] HELD, *Archiv. fur Anat. u. Physiol.*, 1892.
[2] BRAIN, 1895, vol. xviii., p. 37.

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surmised by Loewenthal,[1] who performed hemisection of the posterior part of the medulla of the kitten at the level of the decussation, with the result that there was atrophy of large fibres upon the ventral and external aspect of the pyramid, which he believed did not belong to this structure. Bechterew[2] has shown that a system of fibres in this situation myelinate earlier than the pyramidal fibres. So, too, Bouchard[3] noticed that in man the descending degeneration in the lateral column was greater after injury to the medulla than after injury higher up, and that the medullary fibres were situated at the lateral border of the pyramidal tract. It is clear, however, that, as we have already seen, the fact that there is a constant accession of fibres to the pyramidal system as lower and lower levels are reached, makes it probable that some of the fibres which attracted the attention of these observers were derived from this source, as well as from the system which I am dealing with, for the pyramid was of course injured in the instances from which their observations were derived.

It will, I think, be well to defer the discussion of the direct antero-lateral tract which engaged our attention earlier in the paper, until we have considered the results of cutting out Deiters' nucleus; but before leaving this part of the subject it may be well to emphasise one or two points in connection with this tract. It ought to be clearly remembered that its position is one which does not encroach on the true limits of the direct pyramidal tract to any extent, that its fibres do not extend to any extent along the margin of the anterior median fissure, that it extends throughout the spinal cord to the lowest part of the lumbar region, and that its fibres pass to the grey matter of the anterior horn of the same side, without any evidence of their decussating in the anterior commissure or by any other route. It will be found of extreme importance to keep all these points clearly in mind when we come to further discuss this tract in connection with an apparently similar tract which has been derived from other sources.

[1] LOEWENTHAL, *Rev. Med. de la Suisse Romande*, 1886.
[2] BECHTEREW, "Die Leibungsbahnen," 1894, p. 47.
[3] BOUCHARD, *Archiv. gén. de Méd.*, 1886.

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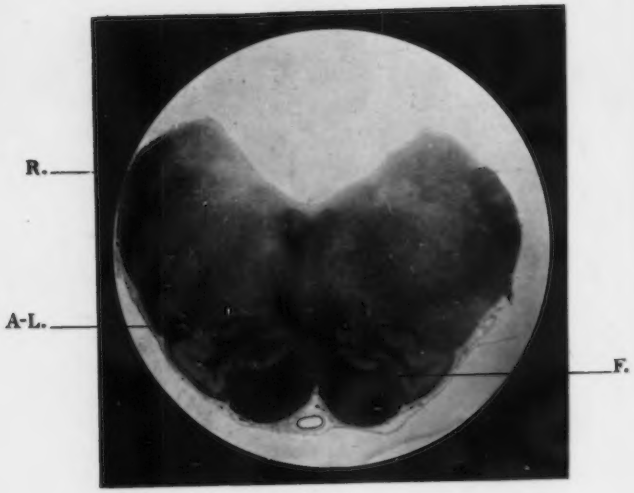
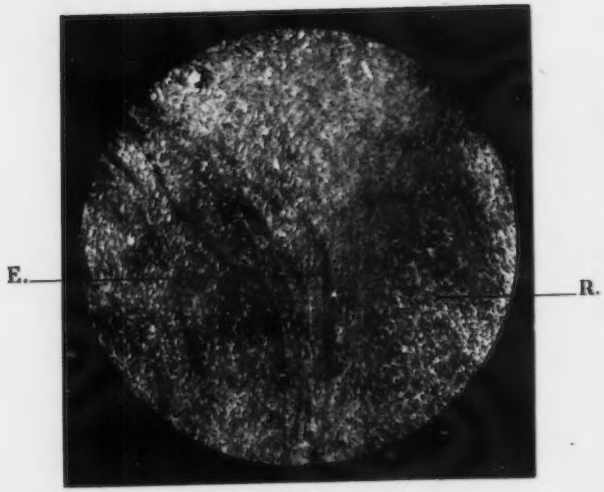


FIG. 5.



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(*b*) *Afferent Tracts.*—There still remains to be described the afferent tracts which were found degenerated after a lesion to the lateral medullary region. Close to the seat of lesion nearly the whole of the periphery of the lateral border of the medulla is occupied by degenerated fibres, but on passing farther towards the brain this more or less complete band of degenerated fibres becomes more and more separated into two distinct groups, until they eventually are no longer connected with each other. The most dorsal of these groups of degenerated fibres occupies the confines of the restiform body, while the ventral group of fibres occupies the position of the afferent ventral cerebellar tract (see fig. 5). The former group naturally passes to the cerebellum in the restiform body, some of the fibres to the middle lobe to end on the same side, and others through this to the opposite side, to end eventually on the ventral aspect of the vermis in the region of the nuclei globosi.

The ventral tract of degenerated fibres passes through the pons as a well defined bundle occupying the region external to the superior olive, and being situated on the ventral side of the emergent root of the seventh cranial nerve, and further forward immediately on the ventral side of the emergent root of the fifth nerve. These degenerated fibres pass round the fifth nerve and course along the inner side of the transverse fibres of the pons (middle cerebellar peduncle), and eventually reach the middle lobe of the cerebellum by turning back over the superior peduncle of the organ.

This tract follows so closely the course which the ventral cerebellar tract has been described as taking, that it is probable that they are identical; accordingly these results confirm the observations of Loewenthal,[1] Mott,[2] Auerbach,[3] and Tooth.[4]

But, in addition to the portion of this ventral cerebellar tract which reaches the cerebellum, another portion has yet

[1] Loewenthal, *loc. cit.*
[2] Mott, Brain, 1892 and 1895.
[3] Auerbach, *Virchow's Archiv.*, Bd. 121, 1890, p. 199.
[4] Tooth, Brain, 1892, p. 397.

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to be described, which lies internally to this more compact bundle, whose course has been already traced, as seen on transverse section at the levels of exit of the seventh and fifth cranial nerves. Though forming a distinct tract of degeneration, these fibres are fewer in number and more scattered than the other bundle, from which they are pretty clearly separated at the level of the exit of the fifth nerve. At this level they are close to the outer end of the fillet, which is otherwise free from degeneration on this side, and in their further course towards the mesencephalon they remain closely associated with this system of fibres. In the region of the posterior corpora quadrigemina they form a well marked though scattered band of degeneration, following more or less the distribution of the fillet, and in the region of the anterior quadrigeminal bodies they are still to be seen as a well marked though scattered tract chiefly occupying the region of the dorsal end of the fillet. Indeed, a few of the most dorsal of these fibres more scattered than their fellows, can be seen coursing inwards towards the middle line, and can be traced to a point a little external to the position of the descending root of the fifth. It will thus be seen that this result is in entire accord with the observations of Mott[1] with regard to this portion of the ventral cerebellar tract, and is quite opposed to the view expressed by Patrick[2] who was unable to find any such degeneration in the anterior quadrigeminal region, and accordingly concluded that Mott's results were probably due to an artifact.

In that the lesion interrupted a considerable proportion of the arciform fibres in the formatio reticularis, well marked degeneration of these fibres passing to the opposite inter-olivary layer was found. On being traced forward through the pons these degenerated fibres were found to occupy both the median and lateral fillet. The degeneration of the fillet is well marked, and can be traced with ease through the mesencephalon to the optic thalamus.

In addition to these arciform fibres which pass to the opposite inter-olivary layer, other fibres are interrupted in

[1] Mott, *loc. cit.*
[2] Patrick, *Journal of Nervous and Mental Diseases*, February, 1896.

their course through the formatio reticularis, notably those passing to the inferior olives, which fibres are, of course, degenerated and can be clearly seen passing to these structures.

Part II.—Degenerations Consequent on Division of the Restiform Body.

The description of the cerebellum, as having three peduncles, appears to me to be erroneous and misleading. The possibility of distinguishing four peduncles by which this organ is connected with contiguous structures is so clear that it is difficult to see why two of these paths of connection should be considered as one. The direct sensory cerebellar tract of Edinger is a structure entirely separate and distinct from the restiform body, and ought to be so regarded. Anatomically they stand out clearly and distinctly as two definite structures, having little if any resemblance, and having connections totally distinct from each other. Embryologically it has been found that the fibres of the direct sensory cerebellar tract receive their medullary covering at a different period to the fibres of the restiform body. Experimentally I have shown that ablation experiments on the cerebellum are followed by degeneration in the medulla oblongata limited accurately to the confines of the restiform body strictly so-called, and as opposed to the direct sensory cerebellar tract which lies to the inner side of the restiform body; and I hope to bring forward further evidence later in this paper in support of the same contention. So, too, in all the experimental evidence there is with regard to ascending degeneration reaching the cerebellum after interruption of afferent tracts in the spinal cord, the degenerated fibres have been accurately limited to the confines of what ought strictly to be looked on as the restiform body, and do not in any way encroach on the area occupied by the direct sensory cerebellar tract of Edinger, or become in any way connected with this tract.

Throughout this paper, then, I shall, in referring to the restiform body, mean the structure "R," shown in fig. 6,

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as opposed to the direct sensory cerebellar tract "E," seen in the same figure lying to the inner side of the restiform body.

Looked at in this way, the cerebellum must be regarded as having four peduncles—an anterior, a middle, and two posterior, or, as I should prefer to regard it, a posterior (restiform body) and an inferior (direct sensory cerebellar tract).

With this conception of the connections of the cerebellum clearly before us, the degenerations consequent on section of the restiform body (posterior cerebellar peduncle) are as follows :—

Of course, a well-marked degeneration of afferent fibres (passing to the cerebellum) follows such a lesion, but with their precise distribution in that organ we are not at present concerned. The points to which I wish rather to direct attention are in connection with efferent fibres, which degenerate after division of the restiform body. These degenerated fibres are strictly limited to the confines of this tract at first, as after ablation experiments on the cerebellum. In passing caudalwards they occupy more and more of the periphery of the medulla, external to the ascending root of the fifth nerve, owing to some of the fibres becoming situated more and more ventrally as lower levels of the medulla are reached. Fibres from this system pass to end apparently in the formatio-reticularis of the same side of the medulla, while others pass to the olive on the same side and to that on the opposite side. No degenerated fibres could be found passing to the spinal cord by this route, an observation which is in harmony with the results of Ferrier and Turner[1] after division of the restiform body. It will thus be seen that the degenerated fibres met with in this system conform as regards distribution in all respects with those which leave the cerebellum by way of the restiform body after ablation experiments conducted on that organ.

Biedl[2], in supporting Marchi's view that a descending antero-lateral tract degenerates in the spinal cord after

[1] *Loc. cit.* [2] *Loc. cit.*

cerebellar lesions, considers that the fact that I met with a few scattered degenerated fibres in the upper cervical region of the spinal cord is in itself evidence against my contention that no such antero-lateral tract, derived directly from the cerebellum, exists. Now, the exceedingly few degenerated fibres which I described as having been met with in the upper cervical cord were only found in some instances, and knowing how easy it is to get slight involvement of Deiters' nucleus in cerebellar lesions, I prefer to look to such a slight accidental complication as accounting for the existence of the insignificant number of degenerated fibres met with in the upper part of the spinal cord. Certainly nothing could be more erroneous than any interpretation of my results of ablation of the cerebellum which finds in them evidence of a descending antero-lateral tract in the spinal cord having its origin in the cerebellum. The contrast between these results and those to be presently described as resulting from injury to Deiters' nucleus is so striking that it is difficult to believe that this tract has its origin in the cerebellum.

Biedl also appears to be under the impression that I denied the existence of fibres degenerating from the cerebellum by way of the restiform body to the opposite inferior olive. The comment in my paper on the subject was that the number of fibres passing to the opposite olive was much smaller than was expected, and did not form so large a tract as that which appeared to have been met with by Ferrier and Turner.

Part III.—Degenerations following Section of the Direct Sensory Cerebellar Tract.

There appears to be little room for doubt that the degenerations met with after section of the direct sensory cerebellar tract are in reality due to the implication of Deiters' nucleus by this lesion. Imbedded as it is, in the fibres of the direct sensory cerebellar tract, it is an almost impossible task to divide this tract without injury to the nucleus. The only way to accomplish division of the tract without injury to the nucleus is to make the incision as

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close to the cerebellum as possible, a procedure attended in its turn by the risk of injury in the cerebellum of structures other than the tract in question. In only one of my experiments am I able to satisfy myself that section of the direct sensory cerebellar tract has been accomplished without apparent injury to Deiters' nucleus (see fig. 7); in some the injury to the nucleus is slight, in others well marked, and belonging to the latter category are the cases in which I deliberately attempted to sever this nucleus from its connections with the medulla. The more obviously Deiters' nucleus was injured, the more marked were the degenerations about to be described, and the slighter the damage to the nucleus the less evident were these degenerations.

In that the only degenerations met with are precisely those which followed the cutting off of Deiters' nucleus from its connections with the medulla, it would be superfluous to describe these now, as they will have to be described in the next section of the paper, which deals directly with lesions of Deiters' nucleus.

No afferent fibres were found degenerating in the direct sensory cerebellar tract between the nucleus of Deiters and the nucleus globosus in the cerebellum, a fact which supports the view of Ferrier and Turner[1] that this is an efferent tract from the middle lobe of the cerebellum to Deiters' nucleus.

Part IV.—The Results of severing the connection of Deiters' Nucleus with the Medulla.

The lesion produced in this case is well shown in fig. 8, from which it will be seen how clean the line of incision was —free from any inflammatory complication and without any damage to neighbouring parts, such as the restiform body or the sixth nucleus, &c. Of the degenerations which result from such a lesion, that which concerns us in the first instance is the degeneration of a tract which leaves the seat of lesion and passes as a well-marked band of degeneration through the formatio reticularis, about midway between

[1] *Loc. cit.*

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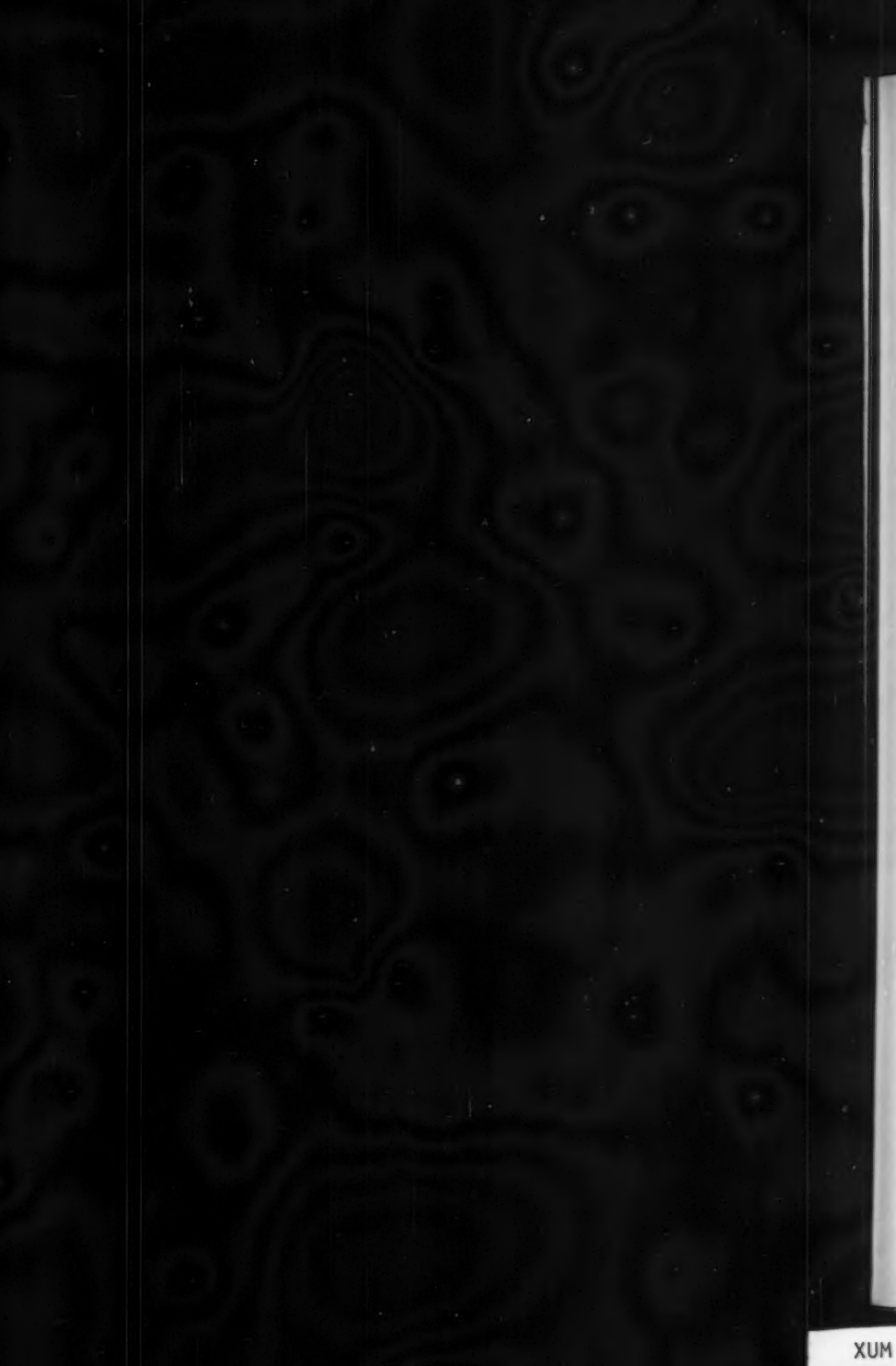

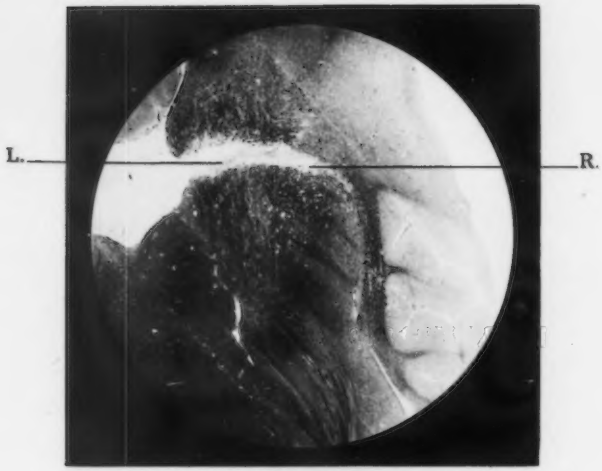

FIG. 7.

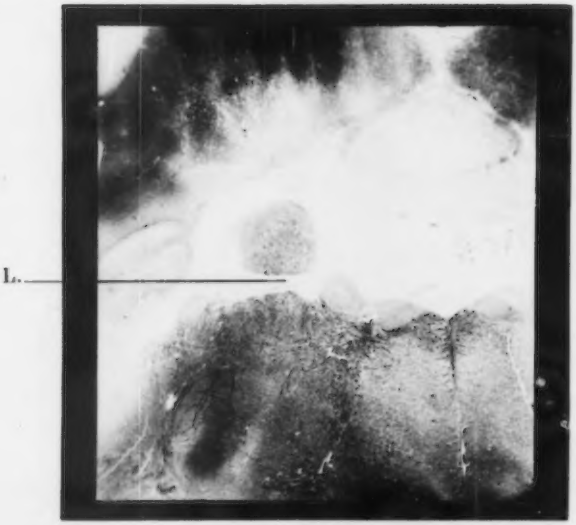

FIG. 8.

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the ascending root of the fifth nerve and the raphe, and which occupies a considerable vertical extent between the dorsal and ventral aspects of the medulla, as seen on transverse section (see fig. 9). In passing caudalwards they are to be found forming a well-marked group of fibres, situated external to the inferior olive and internal to the nucleus lateralis. When the upper cervical region of the spinal cord is reached they are to be seen as a well-marked antero-lateral band of degeneration, occupying the periphery of the cord and extending forward to within a short distance of the anterior median fissure where the band ends; degenerated fibres from this source do not extend to the anterior median fissure, nor do they encroach on the region ordinarily supposed to be occupied by the anterior direct pyramidal tract (see fig. 10). On being traced further caudalwards in the spinal cord, however, this tract becomes blended with another, which we shall subsequently find is derived from fibres which reach the spinal cord by way of the posterior longitudinal bundle on the same side, and these two tracts together form an antero-lateral tract occupying the periphery of the cord, and extending down the margin of the anterior median fissure, thus encroaching on the area of the anterior direct pyramidal tract. The degenerated fibres become much more scattered in the thoracic cord, especially those in the anterior column and those at the periphery of the antero-lateral region of the cord, while still forming a well-marked band, are more scattered, so that the band is broader than that seen in the cervical region. In the lumbar cord there are next to no degenerated fibres left at the periphery of the antero-lateral region of the cord, but a well-marked band of degenerated fibres is situated along the margin of the anterior median fissure.

In their course through the spinal cord these degenerated fibres pass to the grey matter of the anterior horn of the same side. Evidence of the decussation of some of them in the anterior commissure to reach the anterior horn of the opposite side was found.

From certain considerations to be afterwards dealt with, it is probable that the degenerated fibres met with in the

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lumbar region of the cord are derived from this system of fibres which we traced through the formatio reticularis in the medulla, and that none of those which pass to the anterior columns by way of the posterior longitudinal bundles reach the lumbar cord.

It is of interest to note that the degeneration as seen in fig. 10, while occupying the periphery of the antero-lateral region of the spinal cord does not become specially dense close to the anterior tip of the fissure, as was the case after a lesion to the lateral region of the medulla, and shown in figs. 2, 3 and 4.

The tract corresponds in position and general characters to that described by Marchi, as degenerating after ablation of the cerebellum.

Ferrier and Turner[1] failed to confirm Marchi's observation, but found that such a degeneration resulted when Deiters' nucleus was injured. In a previous investigation[2] I also failed to confirm Marchi's observation. It is interesting and instructive to note that while after ablation of one lateral lobe of the cerebellum, there is an entire absence of any degenerated fibres occupying the position of the tract seen in the formatio reticularis after a lesion of Deiters' nucleus, there is nevertheless a well-marked degeneration of fibres in the restiform body and peripheral region of the medulla occupied by fibres derived from that system, a region free from degeneration after injury to Deiters' nucleus. Further, after ablation of one lateral lobe of the cerebellum with well-marked degeneration in the restiform body, an antero-lateral efferent tract in the spinal cord was not found, while a well-marked tract as is to be seen in fig. 10, was obtained after the lesion to Deiters' nucleus, in which the restiform body was free from degeneration.

Mott,[3] when cutting off the posterior column nuclei from the arciform fibres, found that in some instances in which the lesion extended rather far forward and too deeply, causing injury to ground fibres and to some of the ocular nuclei

[1] *Loc. cit.* [2] *Loc. cit.* [3] Mott, Brain, 1895.

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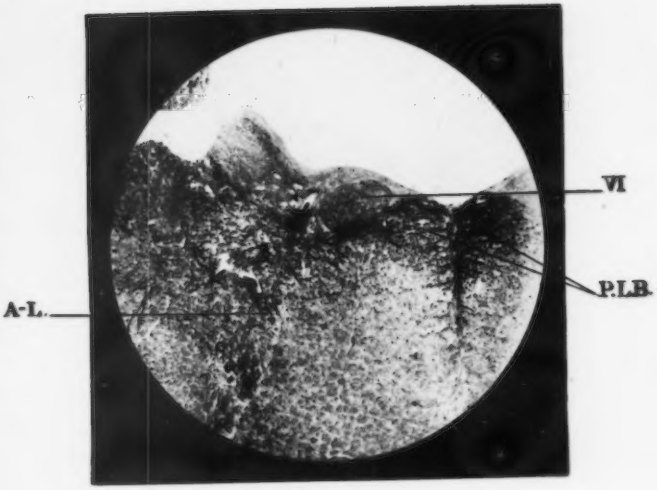

FIG. 9.

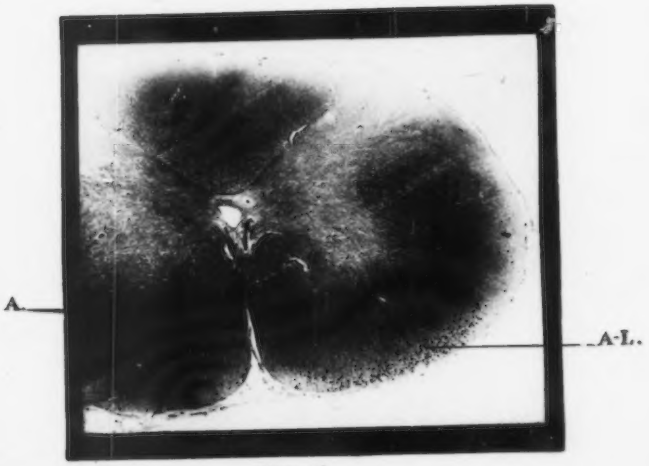

FIG. 10.

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situated in the floor of the fourth ventricle, there was degeneration of an antero-lateral tract similar to that described by Marchi, which passed down to the lumbo-sacral region of the spinal cord, most of the fibres decussating in the anterior commissure to go to the opposite anterior horn.

More recently Biedl,[1] dividing the restiform body in cats, considers that he has completely confirmed Marchi in that he obtained degeneration of this antero-lateral tract in the spinal cord, which he believes is derived from the cerebellum. Further consideration of this antero-lateral tract, about whose origin there is so much difference of opinion, is best deferred until we are in a position to discuss the whole question of what tracts exist in this region of the spinal cord, and what are the most probable sources from which they are derived.

Passing to the consideration of other tracts, which degenerate after cutting out Deiters' nucleus, we find that running inward from the seat of lesion are well marked strands of degenerated nerve fibres which cross the medulla towards the raphe and in their course pass on the ventral aspect the nucleus of the sixth nerve, without any evidence of their entering this nerve or its nucleus at any point (see fig. 9). Some of these degenerated fibres enter the posterior longitudinal bundle of the same side, while others pass across the middle line at the raphe to enter the posterior longitudinal bundle of the opposite side. Having entered the posterior longitudinal bundles, some of these degenerated fibres pass in these structures cephalwards, while others pass caudalwards, so that both an ascending and a descending degeneration results in the posterior longitudinal bundles after a lesion such as that with which we are now dealing. In tracing these degenerated fibres caudalwards it is difficult to say that one posterior longitudinal bundle contains more of them than does its fellow (see fig. 11). When the cervical region of the spinal cord is reached they are found to occupy a position on the ventral aspect of the cord scattered

[1] Biedl, *loc. cit.*

in the anterior columns on each side of the anterior median fissure, and being least scattered in the region of the anterior columns nearest to the anterior commissure and grey matter of the anterior horns. As has already been said, further caudalwards in the cord the fibres of this system on the side of the lesion become intermingled with those of the tract which we traced from the site of the lesion through the formatio reticularis of the medulla to the antero-lateral region of the spinal cord. Judging from the degenerated fibres in the opposite anterior column, which have no other tract of degenerated fibres to become intermingled with, and whose further course through the spinal cord can consequently be followed with much greater certainty, the fibres of this system do not appear to pass far caudalwards in the thoracic cord, but become thinned out and lost. In the lumbar cord no vestige of this tract of fibres can be seen on the side opposite to the lesion, it is, therefore, probable that the anterior tract of degenerated fibres which is to be found in this region of the cord on the side of the lesion is derived solely from the tract traced through the formatio reticularis of the medulla from the seat of lesion.

The fibres which pass to the cord in the posterior longitudinal bundles end in the grey matter of the anterior horn of their own side, and, as far as can be seen, none of these fibres decussate in the anterior commissure to reach the opposite anterior horn.

The degenerated fibres which pass cephalwards in the posterior longitudinal bundles are shown in fig. 12, and can be traced to the quadrigeminal region, where they appear to terminate. All the fibres which pass across the medulla from the seat of lesion do not enter the posterior longitudinal bundles, some of them pass beyond the opposite posterior longitudinal bundle, course along the ventral aspect of the opposite sixth nucleus to end in the formatio reticularis of that side apparently; while others turn forward before they reach the raphe, and seem to terminate in the nuclei of the formatio reticularis on the side of the lesion.

Some degenerated fibres pass from the seat of lesion and arch forward in the formatio reticularis, pass dorsally to the

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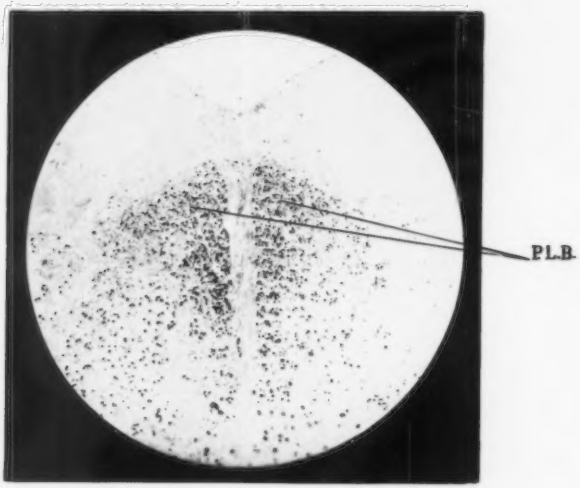

FIG. 11.

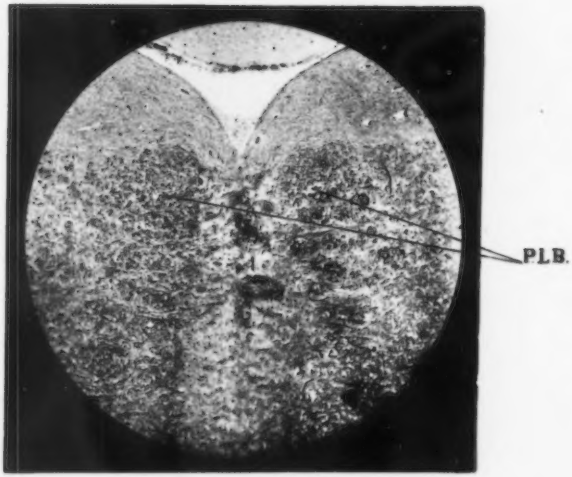

FIG. 12.

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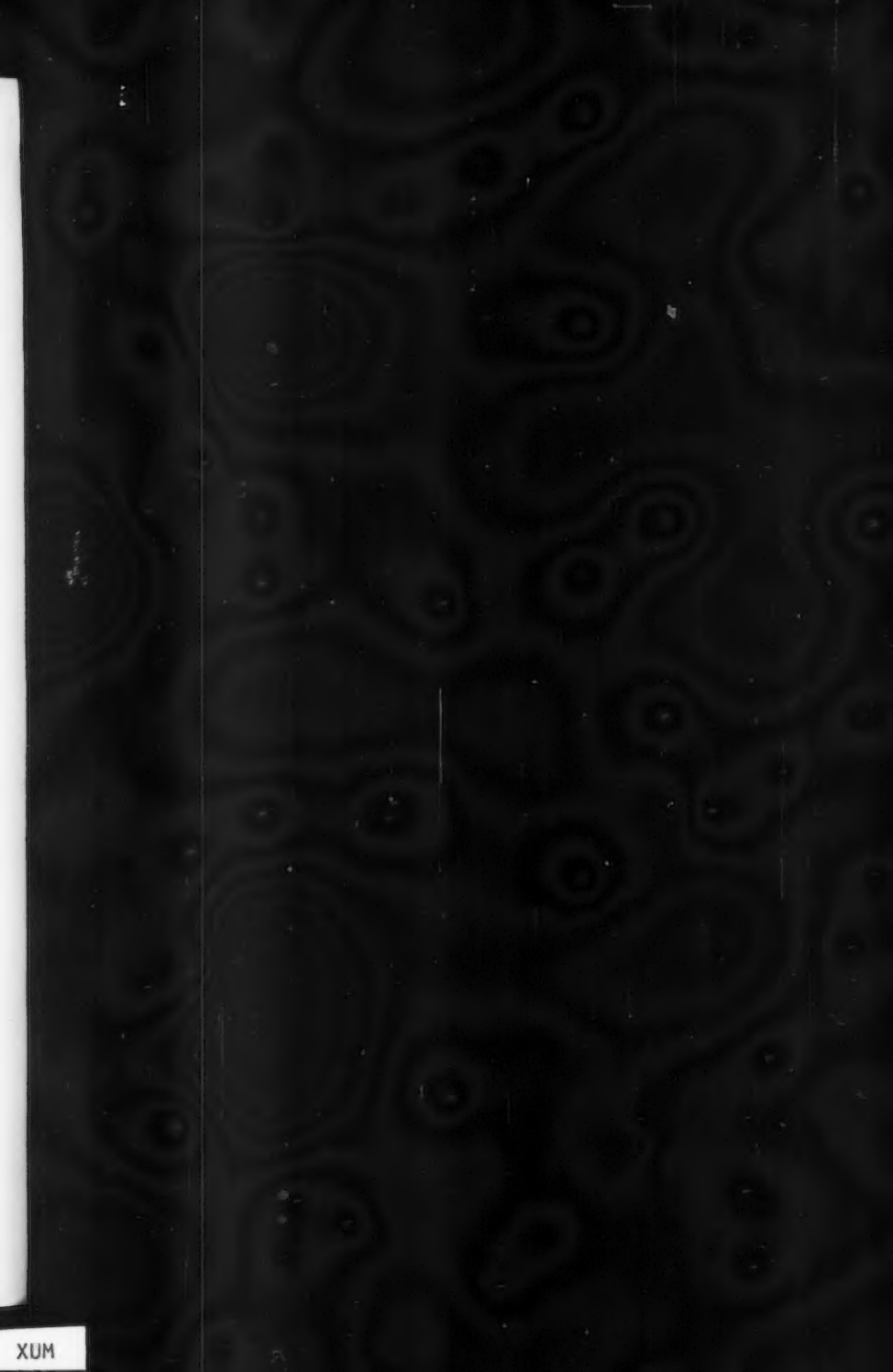

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superior olive of their own side, and the pyramid on both sides to the neighbourhood of the opposite superior olive, some of them appearing to enter this structure.

Other fibres which course through the formatio reticularis in this manner and cross the raphe become intimately associated with the opposite fillet, and pass with this structure to the anterior quadrigeminal region. They occupy the position of both the mesial and lateral fillet. Though many of these fibres which cross the raphe are mingled with the trapezoid fibres, the latter are perfectly intact, and show no evidence of degeneration.

Part V.—Degenerations Resulting from Transverse Section of the Posterior Column and their Nuclei on one side in the Medulla Oblongata.

My object in undertaking this part of the investigation was to exclude the possibility of any of the results obtained after the lesions already described, more especially that in the region of Deiters' nucleus being due to accidental injury to the posterior column nuclei, for it will be remembered that after a lesion in the region of Deiters' nucleus there resulted degeneration of a tract which occupied the position of the fillet on the side opposite to the lesion, and whose fibres followed the course of the fillet through the pons and mesencephalon. To have undertaken this part of the research with any other object would have been superfluous in view of the admirable work that has been done by Mott[1] with regard to the relationship which exists between the posterior column nuclei and the mesial and lateral fillets.

With the exception of such degenerations as resulted from inclusion of part of the restiform body in the lesion, in some cases, the only degenerations which resulted were of fibres entering the cerebellum by the restiform body, and the arciform fibres which passing from the posterior column nuclei course through the formatio reticularis cross the middle line, and thus reach the opposite inter-olivary layer.

[1] Mott, Brain, 1895, p. 1.

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These degenerated fibres pass forward through the pons, occupying the position of the median and lateral fillets, to the mesencephalon. The amount of degeneration of the fillet which results is only slight. This is only what was to be expected when we take into consideration how slight is the damage of the posterior column nuclei produced by a lesion passing transversely through them. So small in amount was the degeneration that it did not appear likely to be profitable to attempt to trace these fibres further forward than the anterior quadrigeminal region of the mesencephalon. The results as far as they go are in accord with those of Mott, Ferrier and Turner, and others. With regard to the additional degenerations present when some part of the restiform body was included in the lesion it is unnecessary to say anything here in that the subject of the degenerations consequent on lesions of the restiform body has already been dealt with. But, apart from this complication, degenerated fibres passed to the cerebellum in the restiform body. No efferent tract was found degenerated in the spinal cord after the lesion to the posterior columns and their nuclei, even when complicated by lesion to part of the restiform body.

(4) Discussion Relating to the Origin of the Direct Efferent Tracts met with in the Spinal Cord.

We are now in a position to discuss the possible or probable origin of the direct tracts met with in the ventral region of the spinal cord, but before doing so, it may be well to briefly summarise the various tracts to be dealt with. They are :—

(1) The antero-lateral tract, said to be derived from the cerebellum.

(2) The antero-lateral tract, derived from the region of Deiters' nucleus.

(3) The antero-lateral tract found degenerated after injury to ground fibres and some of the cranial nerve nuclei.

(4) The anterior column fibres which degenerate after hemisection of the mesencephalon.

The evidence that there exists, at any rate in man, a direct anterior pyramidal tract, is clear; but it is equally clear that this is not the only efferent tract which occupies this, the ventral region of the spinal cord. Less certain is it, however, as to how many additional efferent tracts occupy this region of the cord; nevertheless, the evidence at our disposal on this point suggests the probability that there are at least two such additional systems of fibres derived from very different sources. As to the origin of the one, there is little in dispute. Boyce[1] found that after hemisection of the mesencephalon the lesion passing between the anterior and posterior corpora-quadrigemina, fibres degenerated in both anterior columns, and that those to the opposite anterior column crossed from the seat of lesion to the opposite side of the mesencephalon in Meynert's decussation; and Held[2] states that Meynert's decussating fibres have their origin in the superior corpora-quadrigemina, and take part in the formation of the antero-lateral column. According to Boyce, these degenerated fibres reach the anterior columns of the spinal cord by way of the posterior longitudinal bundles, and cannot be traced beyond the lower part of the cervical region of the spinal cord.

The behaviour of those degenerated fibres which pass to the anterior columns of the spinal cord by way of the posterior longitudinal bundles, after injury to Deiters' nucleus is so similar to that of the fibres described by Boyce, that though taking their origin at different levels of the central nervous system, they nevertheless appear to belong to the same system of "internuncial" fibres.

The other efferent system of fibres which occupy the anterior columns of the spinal cord is a well-marked antero-lateral tract, about the source of origin of which there has been much dispute. Thus Marchi described this tract as degenerating in the spinal cord after ablation of the cerebellum; while my own observations were in accord with those of Ferrier and Turner, who failed to confirm Marchi's observations; these observers, however, found this tract

[1] *Loc. cit.* [2] *Loc. cit.*

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degenerated when Deiters' nucleus was injured. Mott, on the other hand, met with this tract degenerated after injury to ground fibres, and some of the ocular nuclei ; while Biedl, dividing the restiform body 1—1½ cm. above the calamus scriptorius, found this tract degenerated, and believed that his observation confirmed that of Marchi, and concluded that the tract in question is derived from the cerebellum.[1] My own most recent experimental observations on this point are, however, entirely at variance with Biedl's results, for I find that this tract only degenerates when Deiters' nucleus is injured, or severed from its connections with the medulla oblongata. I have elsewhere[2] called attention to the possible errors which may be responsible for Marchi's results, and need not repeat myself in this connection here, except to say that it is my belief that the degeneration of this tract was probably due to accidental circumstances, and that it is probable that such a tract does not degenerate after a lesion absolutely limited to the cerebellum. So careful was I not to injure the medulla or contiguous structures when extirpating portions of the cerebellum, that I preferred to leave a thin layer of cerebellar tissue between my lesion and the medulla, in most instances, rather than run the risk of injury to the medulla. In no instance in which there was no injury to the medulla did I find this tract degenerated. The only possible source, then, from which this tract could be derived is from this fragment of cerebellar tissue left, but I feel convinced that the amount of this was too insignificant to give rise to a tract of such importance. Nevertheless, in a controversy of this kind, in which I am most anxious that the true origin of the tract in question should be decided on, I think it right to put forward all the possibilities of error that occur to me. It is a significant fact, however, that when I removed one lateral half of the cerebellum I obtained a well-marked degeneration of the restiform body, properly so called, and that the degenerated fibres from this

[1] As has already been said, the researches of Thomas also confirm Marchi's observation.

[2] *Loc. cit.*

source occupied the periphery of the medulla at lower levels, and were only seen in the formatio reticularis in their passage to the olives, and possibly to the nuclei of the formatio reticularis, while when I injured the nucleus of Deiters, or severed it from its connections with the medulla, I obtained no such degeneration in the restiform body proper, or in the peripheral region of the medulla, where such a well-marked degeneration existed when the restiform body was degenerated; but, instead, get a well-marked tract of fibres degenerating caudalwards from the region of Deiters' nucleus through the formatio reticularis to the antero-lateral region of the spinal cord.

In attempting to compare my results with those of Biedl, the first possibility that suggests itself to me is that this observer looks upon the restiform body and the direct sensory cerebellar tract of Edinger as one structure, and that what he regards as restiform body I regard as restiform body plus direct sensory cerebellar tract. Let us, therefore, consider what possibility there is that a lesion of the direct sensory cerebellar tract could be made without injury to Deiters' nucleus, which, as we are all aware, is intimately imbedded in this tract. Certainly this object will not be obtained by a lesion dividing the restiform body and ascending root of the fifth nerve 1—1½ cm. above the calamus scriptorius. A far more likely result of such a lesion would be to interrupt any fibres that might be passing from Deiters' nucleus caudalwards, or any derived from the direct sensory cerebellar tract which do not end in Deiters' nucleus (if there are any such fibres), but pass it to reach some region of the medulla caudal to it. The only lesion of the direct sensory cerebellar tract followed by degeneration in the medulla and spinal cord caudal to Deiters' nucleus that can be regarded as indicating that the fibres so degenerated are derived from the cerebellum is the section of the tract between Deiters' nucleus and the cerebellum high enough up, that is, near enough to the cerebellum to avoid injury to Deiters' nucleus. So difficult is this that I have never accomplished the task without some injury to the nucleus in question, except in one instance, despite the fact that I invariably

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attempted to divide the tract as close to the cerebellum as possible without at the same time producing any damage to the cerebellum itself as opposed to this strand of fibres connected with the organ. This much, however, is certain, and that is that the more obvious the lesion to Deiters' nucleus, the more marked was the tract degenerating in the formatio reticularis and passing down the antero-lateral region of the spinal cord, and the less evidence there was of any injury to the nucleus, the less degeneration could be found passing through the formatio reticularis to the spinal cord; indeed, in the one instance in which the nucleus appeared to have escaped injury no sign of degeneration of the antero-lateral or any other tract was found in the spinal cord, so that the evidence seems strong, that the antero-lateral tract originates in Deiters' nucleus, and is not derived directly from the cerebellum.

The observation of Von Monakow,[1] that the cells of Deiters' nucleus atrophy after hemisection of the spinal cord in the cervical region of a new-born rabbit is worthy of careful attention in this connection. Of no less importance are the observations of Held based on the myelination method, for he finds that the antero-lateral ground bundles and lateral limiting layer of the spinal cord are connected with Deiters' nucleus by means of the longitudinal fibre systems of the reticular formation.

But to return to the results obtained by Biedl, we must remember that his lesion was situated at a point 1—1½ cm. above the calamus scriptorius, that it penetrated sufficiently deeply into the medulla to divide the ascending root of the fifth nerve as well as the restiform body, and that as a result of this lesion not only was degeneration of an antero-lateral tract in the spinal cord met with, but also a direct tract in the lateral region of the spinal cord closely associated with the crossed pyramidal tract, as far as position is concerned. Now, I have already shown in an earlier part of this paper that this is precisely the condition of things met with after a lesion of the formatio reticularis, external to the inferior

[1] V. Monakow, *Archiv. f. Psychiat u. Nervenkrank.*, xiv., 1883, p. 1.

olive, and in the region of the nucleus lateralis. Further, I have called attention to the fact that the lateral tract corresponds closely with a similar tract described by Boyce as degenerating after hemisection of the mesencephalon in the region between the anterior and posterior corpora quadrigemina.

With such evidence before us it is impossible to escape from the supposition that Biedl's lateral tract is in reality none other than that met with by Boyce and myself after the lesions just described, and that the degeneration resulted from its interruption in the formatio-reticularis. A reference to Boyce's, fig. 4, p. 329, will show that his so-called lateral columnar tract is so closely related in position to the ascending root of the fifth that any lesion to the latter structure would be more than likely to also involve these lateral columnar fibres, and would account for the degeneration met with by Biedl in the lateral column of the cord.

The antero-lateral tract of Biedl in all probability results from interruption of fibres passing from Deiters' nucleus in the formatio reticularis, or possibly from injury to this nucleus itself. That this is the probable origin of the degenerated fibres met with in this tract, and that it does not simply indicate an interruption of Boyce's anterior columnar-fibres is shown by the fact that the fibres traced by Boyce to the anterior columns of the cord pass in the posterior longitudinal bundles well out of reach of Biedl's lesion, and that the latter observer describes degenerated fibres in the formatio reticularis in precisely the position in which they have been met with by me after lesions of Deiters' nucleus.

It must not be lost sight of in this discussion that however much difference of opinion there may be with regard to the origin of the antero-lateral efferent tract, it is the only one met with by me after complete section of the restiform body proper together with the direct sensory cerebellar tract, and that absolutely no evidence of any direct lateral tract situated near the crossed pyramidal tract was met with. Mott's result is of importance in this connection, for his lesion involved the sixth nucleus chiefly,

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and would thus almost certainly interrupt the fibres passing from the region of Deiters' nucleus to the antero-lateral region of the cord; and in accordance with my results he met with an antero-lateral tract only, and with no lateral tract in the region of the crossed pyramidal tract.

(5) General Conclusions.

The results obtained in connection with the various experimental lesions that have been described in this paper, justify the following conclusions:—

(1) The descending antero-lateral tract which degenerates in the spinal cord after a lesion of the lateral region of the medulla is probably the same as that met with after injury to Deiters' nucleus, such slight differences as exist between the two tracts being possibly accounted for by the fact that some of the fibres which degenerated after the lesion to Deiters' nucleus may have escaped injury in connection with the lesion to the lateral region of the medulla. This tract is probably identical with that described by Marchi, as degenerating after lesions of the cerebellum; Mott, after injury to ground fibres and some of the cranial nuclei; and Biedl, after section of the restiform body and ascending root of the fifth nerve; its real source of origin being Deiters' nucleus as contended by Ferrier and Turner.

(2) The degenerated fibres which reach the anterior columns of the upper portion of the spinal cord through the posterior longitudinal bundles after a lesion of Deiters' nucleus, are quite distinct from the above tract, and probably belong to some system of inter-nuncial fibres similar to those traced by Boyce to the anterior columns of the spinal cord, by way of the posterior longitudinal bundles, after hemisection of the mesencephalon.

(3) The direct descending tract of degenerated fibres met with in the spinal cord, in close relationship to the fibres of the crossed pyramidal tract, after a lesion of the lateral

region of the medulla, is probably identical with a similar tract described by Boyce, after hemisection of the mesencephalon, and by Biedl after section of the restiform body and ascending root of the fifth nerve, the proximity of Boyce's columnar tract to the latter structure accounting for its probable inclusion in Biedl's lesion. The fact that Boyce only traced these columnar fibres to the cervical region of the cord, while I was able to trace them throughout the whole length of the cord, probably indicating that the system of fibres is one to which new fibres are constantly being added as levels more and more caudally situated are reached in passing through the pons and medulla. On this hypothesis, there is nothing peculiar in Boyce's fibres derived from the mesencephalon terminating in the cervical cord, while my interruption of the tract in the lower region of the medulla resulted in degeneration of fibres to the lumbo-sacral region of the cord. The most important objection to looking on Boyce's tract, and that which I have described as belonging to the same system of fibres, is the fact that Boyce's fibres originate in a decussating system, while there is no evidence that the tract I have met with decussates at any point in its course.

(4) That the direct tract in the region of the crossed pyramidal tract is in no way derived from the pyramids is abundantly proved by the fact that in my lesions both pyramids were left absolutely intact, not the slighest injury to either of the structures being detected on most careful microscopical examination.

(5) Fibres derived from the restiform body proper and degenerating caudalwards, after section of this structure, occupy the lateral peripheral region of the medulla in passing further and further caudally, do not form a descending tract in the spinal cord, but pass to the formatio reticularis, and to both inferior olives.

(6) The "direct sensory cerebellar tract" of Edinger is a totally distinct structure from the restiform body, and ought not to be confounded with it. This tract is not an afferent tract, but on the contrary all the evidence obtained on this question points to its being an efferent tract from the nucleus

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globosus of the cerebellum to Deiters' nucleus in the medulla, as has been contended by Ferrier and Turner.

(7) Fibres which degenerate after section of this tract (with injury to Deiters' nucleus) have a different course in the medulla oblongata to those derived from the restiform body.

(8) There is an indirect efferent path from the cerebellum to the spinal cord, through the "direct sensory cerebellar tract" of Edinger and Deiters' nucleus.

(9) One of the tracts met with after a lesion of the lateral region of the medulla corresponds so closely in position and distribution with the afferent antero-lateral tract of Gowers, that it is highly probable that they are identical, and accordingly that the observations of Mott and of Auerbach are correct in this connection.

(10) Another afferent system of fibres which degenerates after the lesion of the lateral region of the medulla, and which is related to the fillet in its course to the quadrigeminal region, is probably the same as that described by Mott as a distinct part of the antero-lateral tract of Gowers, and is certainly no artifact as has been suggested by Patrick.

(11) After injury to Deiters' nucleus, a tract of degenerated fibres can be traced in close association with the opposite fillet to the anterior quadrigeminal region.

(12) So, too, degenerated fibres forming an afferent system pass in both posterior longitudinal bundles to the quadrigeminal region of the mesencephalon after injury to Deiters' nucleus.

(13) None of the degenerations found after section of Edinger's direct sensory cerebellar tract can be ascribed to the severance of fibres of this tract, but all appear to depend on concomitant injury of Deiters nucleus.

(14) Injury of the posterior column nuclei is responsible for none of the degenerations met with in this inquiry, with the exception of a well-marked degeneration of the arciform fibres to the opposite inter-olivary layer with consequent well-marked degeneration of the fillet and a degeneration of fibres passing in the restiform body to the cerebellum.

DESCRIPTION OF FIGURES.[1]

FIG. 1.—Lesion of the lateral region of the medulla oblongata (L) produced by means of a needle. Degenerated fibres, stained black, are seen in the neighbourhood of the lesion at the periphery of the medulla, and also in the fillet (F) immediately dorsal to the opposite pyramid. The inferior olive (O) and both pyramids (P) are absolutely intact.

FIG. 2.—The efferent tracts which degenerate after a lesion such as that represented in figure 1, and which form a continuous band in the cervical region of the spinal cord, from which region the photomicrograph was taken. Two chief areas of degeneration, the one situated ventrally (A-L) and the other close to the crossed pyramidal tract (L.C.), are united by a narrow band of degenerated fibres which occupy the periphery of the antero-lateral region of the cord.

FIG. 3.—A photomicrograph of a section from the thoracic part of the same cord as that whose cervical region is shown in figure 2. The degenerated fibres are more scattered, and the ventral (A-L) and lateral (L.C.) tracts are no longer so continuous with each other as was the case in the cervical region.

FIG. 4.—The two efferent tracts are seen completely separated from each other in the lumbar region of the same cord from which figures 2 and 3 were taken.

FIG. 5.—The afferent tracts which degenerate after a lesion such as that shown in figure 1. Two dark areas of degenerated fibres are seen at the periphery of the medulla, the one irregularly circular (A-L) situated just outside and dorsal to the inferior olive, and the other forming a band which occupies the position of fibres which pass to or from the restiform body (R). F = Fillet.

FIG. 6.—A photomicrograph of a section showing the restiform body (R) with its fibres divided transversely, some of them being degenerated in consequence of a lesion of this structure caudal to the point shown in the figure, and the direct sensory cerebellar tract of Edinger (E) with its fibres undegenerated and cut longitudinally.

FIG. 7.—The line of incision (L) passing through Edinger's direct sensory cerebellar tract is shown in this figure. The dorsal part of the restiform body (R) external to Edinger's tract has been slightly injured, but all other adjacent parts of the medulla and cerebellum have escaped injury.

FIG. 8.—The lesion (L) in a case in which the nucleus of Deiters was severed from its connections with the medulla oblongata.

FIG. 9.—A photomicrograph of the tracts of degenerated fibres which result from a lesion such as that represented in figure 8. One darkly stained band of degenerated fibres is seen occupying a considerable area of the formatio reticularis midway between the ascending root of the fifth nerve and the raphe (A-L). Other degenerated fibres are seen passing on the ventral aspect of the sixth nucleus (VI.) to reach the posterior longitudinal bundles (P.L.B.)

[1] The figures are from photomicrographs, some of which were taken for me by Dr. E. S. Worrall, and others by Mr. L. B. Fleming.

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FIG. 10.—A photomicrograph to show the distribution of the degenerated fibres in the upper cervical region of the spinal cord after the lesion shown in figure 8; they occupy both anterior columns (A), and the periphery of the antero-lateral region of the cord on one side (A-L) as well.

FIG. 11.—The efferent fibres which degenerate in the posterior longitudinal bundles (P.L.B.) after the lesion shown in figure 8.

FIG. 12.—The afferent fibres which degenerate in the posterior longitudinal bundles (P.L.B.) after the lesion shown in figure 8.

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AN EXPERIMENTAL INVESTIGATION OF THE DIRECT PYRAMIDAL TRACT.[1]

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Introduction.

Before entering upon the subject of my paper, I wish to express my thanks to Professor Vaughan Harley for allowing me to carry out this investigation under favourable circumstances at the Laboratory of Pathological Chemistry at University College, and to Mr. Victor Horsley for his assistance, especially during the earlier part of the research, and for the kind and valuable criticism which he was always willing to afford.

The fibres of the crossed pyramidal tract are well known to conduct impulses from the cerebral cortex of one side to muscles upon the opposite side of the body. It has been the object of the present research to determine to what extent the same statement is true for those motor fibres which, at the decussation of the pyramids in the medulla oblongata, do not cross to the opposite side of the cord, but continue on the same side under the name of the "direct pyramidal tract."

[1] The expenses of this research were defrayed by the British Medical Association.

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Method of Investigation.

Animals employed.—Inasmuch as the direct pyramidal tracts form a larger part of the cord in monkeys than in lower animals (and the difference is yet more marked in the case of the *anthropoid* apes), I had hoped and attempted at the outset to carry out this investigation in monkeys. But the necessary severity of the operations and the consequent shock were so great that these animals could not live to the end of the experiment. The same remark applies to cats and to rabbits. The results obtained, therefore, in the present research, are to be taken as applying to dogs only.

In a preliminary note in the *British Medical Journal* of July 18, 1896, allusion was made to an experiment on a monkey which apparently gave positive results. Microscopical examination of the medulla oblongata in that case, however, showed the experimental division to be incomplete.

The following was the method of research:—

(*a*) The dogs were anæsthetised with ether in all cases except the first, when chloroform was used. In no case was the animal allowed to recover from the anæsthetic.

(*b*) A preliminary tracheotomy was performed in every case.

(*c*) The skull was opened over both excitable regions of the cerebral hemispheres without opening the dura mater. All hæmorrhage being arrested, the flap of skin was replaced in order to keep the cortex warm during the progress of the ensuing part of the experiment.

(*d*) The arches of the atlas and axis were exposed by a median incision and removed. By means of a small trephine, an aperture was made in the thin plate of the occipital bone covering the vermis, and this aperture was enlarged and made continuous with the foramen magnum by removing the bridge of bone, which separated the two.

(*e*) The dura mater thus brought to view was opened in its whole length, and the cerebellum, lower part of the medulla oblongata, and uppermost end of the spinal cord well exposed. The fourth ventricle was then carefully opened. The posterior median fissure could now be seen

extending down the cord from the point of the calamus scriptorius, with the columns of Goll standing out prominently on each side of it. The cerebellum was raised slightly by means of an aneurism needle and the lower part of the fourth ventricle exposed to view. A von Graefe's cataract knife was then inserted in the middle line just above the point of the calamus scriptorius, and thrust forward and upward towards the anterior surface of the medulla. The incision thus begun was carried in the middle line down the whole length of the medulla, and part of the spinal cord exposed, care being taken that the point of the knife should not quite reach the anterior or ventral surface of these structures. This part of the operation was then completed by withdrawing the knife and inserting into the incision a blunt flat seeker and pressing it against the posterior surface of the basilar portion of the occipital bone, and the posterior surfaces of the bodies of the vertebræ. This proceeding was found to be necessary because, in those cases where one was fortunate enough to strike the middle line in front, the basilar artery became longitudinally divided if the knife alone was used throughout.

(*f*) At this stage the animal ceased breathing, but was restored by performing artificial respiration for about ten minutes.

(*g*) Attention was now again turned to the cortex cerebri. This was exposed by again reflecting the flap of skin made at the earlier part of the experiment, and opening the dura mater over each hemisphere. Flaps of dura mater were formed and turned upward, so as to expose on each side the crucial sulcus and neighbouring gyri.

(*h*) With a weak faradic current, the excitability of the cortex was ascertained by stimulation of the facial area. Satisfied on this point, one next proceeded to systematically stimulate the motor area in all its parts, and to note what movements were obtained as a result of such stimulation.

(*i*) Finally, the animal was killed, and its brain removed with the upper part of the spinal cord for subsequent examination. The relative accuracy of the division of the decussation was ascertained by macroscopical examination

of the medulla at the time and by subsequent microscopical examination of transverse sections of the same.

Experiments.

In the following list of experiments I make no reference to those in which, owing to death of the animal from hæmorrhage, shock, &c., inexcitability of the cortex, or very incomplete division of the decussation, the result was valueless. Unfortunately, owing to the many difficulties of the investigation, much of my work was lost by accidents of this nature.

No. 1.—Mesial Section.

In this case, the incision passed longitudinally down the left pyramid and divided all the fibres going from the right hemisphere to the left half of the cord; while the division of those fibres which passed from the left hemisphere to the right side of cord, was incomplete.

Movements obtained.—Flexion of the opposite hip was obtained from stimulation of either cortex.

Flexion of the right fore-limb at the shoulder was obtained from stimulation of the left hemisphere: but no movements could be obtained in the left fore-limb by stimulation of either cortex.

No movements of the trunk could be obtained, nor were there any movements on the same side of the body as the excitation.

No. 2.—Mesial Section.

The incision was absolutely in the middle line completely dividing the decussation.

Movements obtained.—Flexion of the opposite hip was obtained from excitation of either cortex. It was equal on the two sides.

There were no movements of the fore-limbs, of the trunk, or of the same side of the body as the excitation.

No. 3.—Mesial Section.

The incision passed down the outer side of the left pyramid. It divided all the fibres going from the right

hemisphere to the left side of the cord, but apparently few of those going from the left hemisphere to the right side of the cord.

Movements obtained.—Flexion of the opposite hip was obtained on excitation of either cortex.

Flexion of the right fore-limb at the shoulder was obtained on stimulation of the left cortex.

No movements of the left fore-limb were obtained, or of the trunk, or of the same side of the body as the stimulation.

No. 4.—Mesial Section.

The incision passed down the outer side of the left pyramid, and was such that very few motor fibres were divided.

Movements obtained.—Powerful movements were obtained in all four limbs. I include this faulty experiment, because it not only serves as a control observation, but also because the tail deviated to the same side as the cortical excitation at a spot just above the leg area on each side (*vide Experiment No.* 6).

No. 5.—Mesial Section.

The incision was median and divided the decussation completely.

Movements obtained.—Flexion of the hip, ankle, knee and toes of each side resulted from stimulation of the opposite cortex.

No movements were obtained of the fore-limbs, of the trunk (including the tail), or of the same side of the body as the cortical excitation.

No. 6.—Mesial Section.

The incision passed very slightly to the right of the middle line and divided the decussation completely, except for a few fibres which passed from the left cerebral hemisphere to the right side of the cord at the lower end of the incision.

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Movements obtained.—On stimulation of the left cortex a slight movement of the right fore-limb digits was obtained. This was, however, soon lost.

Flexion of the hip, knee, ankle and toes was obtained on stimulation of the opposite cortex on each side.

The tail deviated to the same side as the excitation.

No trunk movements were obtained.

No. 7.—Mesial Section.

The incision was median and completely divided the decussation.

Movements obtained.—The only movements obtained in this animal were slight flexion of the toes on each side on excitation of the opposite cortex; a deviation of the tail to the same side as the excitation. The deviation of the tail was more marked to the left than to the right side.

No movements were obtained of the fore-limbs, or of the trunk, or on the same side as the excitation, with the exception of the tail.

No. 8.—Mesial Section.

The incision was median and completely divided the decussation. At the lower end, however, it deviated to the right and apparently divided nearly all, if not all, of the fibres of the direct pyramidal tract on that side.

Movements obtained.—Slight flexion of the toes was obtained on the right side, but no movements of the left, on excitation of the opposite cerebral cortex.

The tail deviated to the left on stimulation of the left cerebral cortex. No deviation to the right could be obtained.

No movements were obtained of the fore-limbs, or of the trunk on either side.

No. 9.—Mesial Section.

The incision was median for the greater part of its length, but deviated slightly towards the left at the upper end of the decussation, and a few fibres at the upper end of the decussation passing from the left cerebral hemisphere to the right half of the cord escaped division.

Movements obtained.—Excitation of the left cortex gave well-marked flexion of the right hip and knee, slight flexion of the left hip, arching of the trunk laterally with the concavity to the left side and deviation of the tail to the right.

Excitation of the right cortex gave slight flexion of the left hip, knee, ankle and toes, and deviation of the tail to the left.

No movements of the fore-limbs were obtained.

No. 10.—*Mesial Section.*

The incision was oblique so that it deviated slightly to the left at the upper end, while at the lower end it encroached slightly on the right direct pyramidal tract. A few fibres passing at the upper end of the incision from the left hemisphere to the right side of the cord escaped division.

Movements obtained.—Excitation of the left cortex gave slight flexion of the hip, knee and ankle on the right side, and contraction of the right back muscles so that the trunk was arched laterally with its concavity to the right.

Excitation of the right cortex gave extension of the hip, knee and ankle on the right side. No movements were obtained on the left side.

There were no movements of the fore-limbs.

No. 11.—*Mesial Section.*

The incision was median and the decussation was completely divided.

Movements obtained.—Flexion of the hip, knee, ankle and toes was produced on each side by excitation of the opposite cerebral cortex, while the tail deviated to the same side as the excitation.

There were no movements either of the trunk or of the fore-limbs.

No. 12.—*Mesial Section.*

The incision was median and completely divided the decussation.

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Movements obtained.—The results were precisely the same as those in the last experiment, except that no movements of the tail were obtained.

Remarks.

There is considerable uniformity in the results obtained in the above experiments, although No. 9, and in part No. 10, appear to be anomalous in many respects.

In cases 2, 5, 7, 11, and 12 the incision precisely carried out the desired conditions. No. 8 went farther, inasmuch as the only tract left intact was the direct pyramidal tract of one side. In none of these cases was any movement obtained either in the fore-limbs or in the trunk. On the other hand, flexion was obtained, on the opposite side to that of the excitation, at the joints of the hind-limbs in all the cases—hip, knee, ankle, and toes in three, toes only in two, hip only in one.

In three of these cases there was deviation of the tail to the same side as the excitation. Experiments 4 and 6 are also in accordance with this observation; while in the anomalous No. 9 the tail deviated to the opposite side.

In all the other experiments, except No. 4, the crossed pyramidal tract was completely cut off from one side of the body only; and in all these cases the results for that side are in accordance with the above observations.

The movements obtained in the limbs were in no case as powerful as those in a dog where the pyramids are untouched. That this is not merely due to shock is shown by facts observed in No. 4 and in several of my unpublished cases. In some of these the medulla was longitudinally divided; but the pyramids were absolutely untouched. In all such cases the movements of the limbs, obtained on excitation of the cortex, were quite powerful.

No importance is to be attached to the fact that flexion rather than extension of the limbs was so uniformly obtained; because the former movement is so much more readily brought about by excitation of the cortex than the latter.

Conclusions.

(1) The number of fibres (channels) in the direct pyramidal tract does not appear to be the same for all individuals of the same species.

(2) Nearly all the fibres of the so-called direct pyramidal tract ultimately cross to the opposite side of the cord.

(3) Its fibres convey some of the impulses from the cortex cerebri to the hind-limb of the opposite side.

(4) A few fibres remain on the same side of the cord to supply the tail muscles of that side.

(5) The direct pyramidal tract conveys no impulses to the fore-limbs or to the trunk.

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A METHOD OF EXAMINING FRESH NERVE-CELLS; WITH NOTES CONCERNING THEIR STRUCTURE, AND THE ALTERATIONS CAUSED IN THEM BY DISEASE.

BY JOHN TURNER, M.B.

Assistant Medical Officer, Essex Lunatic Asylum.

EVER since Nissl introduced his method of staining the chromophilic material of the nerve-cell by methylene blue, considerable doubts have been entertained, at least by English histologists, as to whether the appearances observed were not artificially produced during the somewhat severe processes through which the section had to pass in preparing and staining. Dr. Robert S. Cook,[1] by a modification of the method in which neither alcohol or heat was employed, did much to remove the doubts entertained as to the genuineness of the chromophilic material considered as a constituent of the normal cell structure; still, his method involves the freezing of the cell and its fixation in osmic acid, &c. Recently, Dr. Mott has expressed the opinion that the rod-like and granular arrangements seen in the cell in Nissl's preparations might be due to coagulation by the alcohol, &c., of the albuminous principles of the cytoplasm. That, however, the appearances here referred to are quite genuine, and exist as such in the dead cell, unaltered by reagents, can with certainty be shown by examining fresh cells in the following way.

A small and thin slice of cortex is placed direct in a 0·5 per cent. watery solution of methylene blue (Grubler, B.x.), and is left from three to twelve hours; three hours is sufficient, but the pieces do no harm after twelve hours' soaking.

[1] Eighth Annual Report, New York State Hospital, 1894.

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FIG. 1.

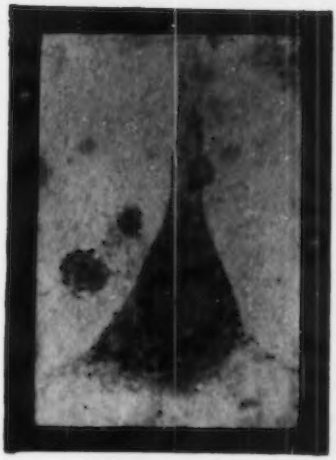

FIG. 2.

FIG. 3.

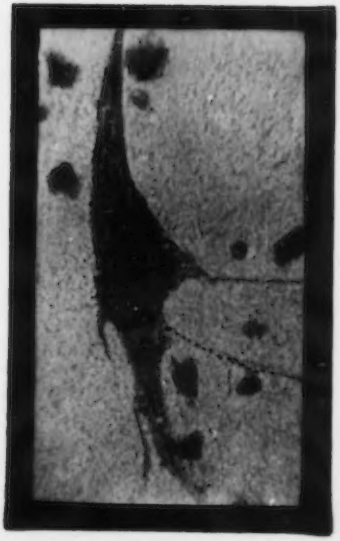

FIG. 4.

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When ready a minute portion is removed with a scalpel from the surface of the piece, through the whole depth of the cortex, the smaller the piece the better. The fragment is laid on a slide and covered with a drop of Farrant's solution (the Farrant is not essential—water suffices—but with the former the preparations keep longer), and covered with a cover-glass, and gentle, uniform pressure, made with two mounted needles, to spread out the fragment, so that its several parts maintain, as far as possible, their relative position to one another.

When the film is sufficiently thin to transmit light, the slide is removed to the stage of the microscope, and further flattening out done with a mounted needle, watching the cells meanwhile through a half-inch objective. Remarkably beautiful preparations of nerve-cells are thus obtained, which are best viewed by artificial light. As a rule these preparations do not keep more than ten days, but they can be preserved for a few days longer, and rendered sharper, if the film is passed to and fro through a flame till the slide is just too hot to bear on the back of the hand. By this means the colour in the cells deepens, and that in the matrix fades quicker than it does if left unheated. Occasionally, for what reason I know not, the films will keep for a long time. (Fig. 2 was photographed from one which had been prepared two years previously.)

These preparations are at their best a few days after first made, as by that time nearly the whole of the blue colour (pink by artificial light) of the matrix has faded, and the blue cell stands out sharply against an almost colourless background.

By the above means one is enabled to study nerve-cells in their original condition, and unaltered by any re-agents (beyond the artificial colouring). A true estimate of their size is formed, and the intimate structure of the cytoplasm is clearly seen. Pigmentation, when present, is distinctly shown; it is generally of a light yellowish green colour, or of a light brownish red. Sometimes the cells (especially the giant pyramids of the motor region) are enormously distended with it, so that they bulge out to more

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than their original size, the collection of pigment being larger than the rest of the cell. The pigment is most usually deposited at the base or along one side of the cell, but it may intervene between the body and apex, apparently blocking off completely the former from the latter.

The nucleolus is darkly stained and sharply defined; the nucleus does not show except in certain pathological conditions. One observes the great elasticity of the cells; with pressure on the cover-glass, their long flexible processes bend about in all directions. The body spreads out and withstands considerable squeezing, returning to its former shape when the force is removed. In senile cells, and those altered by disease, this elasticity is often lost, and very slight pressure will part the processes from the body and cause the latter to break up.

The shape of the cells is seen to be very different in different cases, those from young subjects, &c., are angular with 7, 10 or more processes, while those from the old or degenerated are globular and with few or perhaps none. I am speaking now of the pyramidal cells of the third and fourth layers of frontal and motor cortex, and the large scattered pyramids of the occipital cortex.

Lugaro[1] refers "to the condition which has been termed sclerotic degeneration, in which the cell appears shrunken, intensely stained, and homogeneous," and this he considers "artificially produced by the action of hardening agents under circumstances not well understood."[2] However, that this appearance and condition does occur in certain pathological states, and is not due to artificial means is evident, as such cells are met with by the present method; they generally appear small and badly shaped, and are tough, very considerable pressure on the cover-slip being required to break them down.

The chromophilic material exists normally in the body of the cell in the form of short, thick rods, triangular and spindle-shaped bodies, and in the more interior parts of

[1] *Rivista di Pathologia Nervoso E Mentale*, Aug. 1896.
[2] Quoted from Dr. F. Robertson's Abstract, *Journ. Mental Science*, July, 1897.

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FIG. 5.

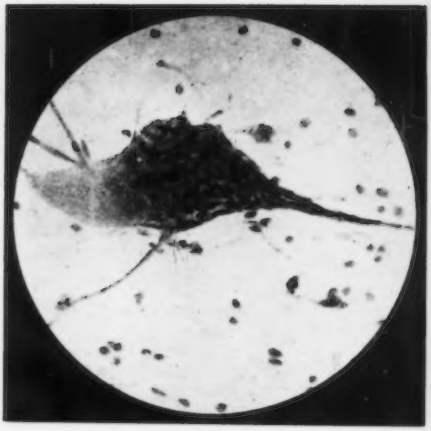

FIG. 6.

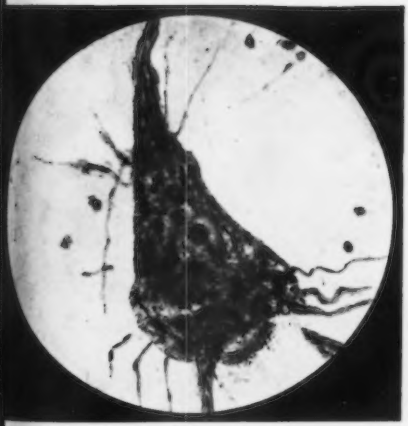

FIG. 7.

FIG. 8.

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the cytoplasm as irregular ill-defined masses; in the apex and processes as somewhat slender, often spindle-shaped threads with their long diameter, corresponding to that of the process. These threads are from 10-20μ long. Such an arrangement is found among the cells (presumably in this respect normal) in the brains of those dying with recent insanity; and to a less extent in those whose mental symptoms have been of longer duration, but in which gross degenerative changes have not taken place (see figs. 1, 5, 7, 9).

An early indication of morbid change is the breaking up of the threads in the processes into a series of small granules or cubes, which, however, are still linearly disposed (see figs. 3, 4 and 10).

Aggregation of several particles into large masses is also apparently an early change (see fig. 11), at least it is found in cells which in other respects appear healthy.

In senile and certain other prolonged degenerative conditions (*e.g.*, advanced general paralysis) it is common to find the chromophilic material arranged in the form of rather large irregular granules distributed sparsely throughout the cell and in its processes, in which latter are no longer any threads to be seen (fig. 12).

In advanced general paralysis one sometimes (probably usually) meets with a complete disappearance of the chromophilic material; film after film from different parts of the cerebrum may be examined, all with a negative result. In these cases the cells often stain rather deeply and diffusely, and are tough and difficult to break. They are, except that often there is no apparent shrinking, in the condition that Lugaro refers to as sclerotic.

Sometimes with general disappearance of the chromophilic material the cytoplasm stains lightly and homogeneously, and is exceedingly fragile, so that it is almost impossible to spread out the film without fracturing processes, and often destroying the contour of the cell. In such cases one generally finds the nucleus well defined, rather deeply stained, round, oval, or triangular, with rounded edges; but although it may be found to have a shrunken appearance in fresh Purkinje cells, it is rarely if

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ever so seen in the cells of the cerebrum. The appearances just described are sometimes found in advanced general paralysis (fig. 8).

In the Purkinje cells of the cerebellum the chromophilic material is scattered uniformly throughout the cytoplasm, in the form of short rods, triangles, or granules, but in certain morbid conditions it is common to find an aggregation of it around the nucleus, while the remainder of the cell contains little or none. This appearance has been described by Dr. Fleming in the multipolar cells of the anterior cornua in rabbits and dogs after ligature of the sciatic nerve, etc.[1]

The processes stain either very faintly or not at all except with certain pathological conditions; thus, those of senile cells, stain often densely.

The cerebellum on account of the more or less uniform size of its elements is a favourable site from whence to obtain specimens for a comparison of fresh and hardened cells.[2]

The shrinking of the Purkinje's cells and their processes in alcohol varies a great deal; in the case of those in which presumably no marked degenerative processes have taken place it amounts to about one-third of the original bulk,

[1] BRAIN, vol. xx., Nos. 77-78.

[2] The following modified method of Nissl I have found gives good results, and as it is far simpler than the original process, it may be worth while to describe it: Small, thin (4—5mm. thick) slices of fresh brain are placed in absolute alcohol, where they remain 24-36 hours, care being taken that each lies perfectly flat and not in contact one with another. The alcohol should be changed twice or three times as soon as it becomes turbid. The pieces are then trimmed so as to be no thicker than 3 mm., and placed in a 2% watery solution of methylene blue B.x. They remain in the solution all night, standing on the paraffin bath, which is kept at 56°—58° C. The following morning the pieces are removed from the staining fluid and put into absolute alcohol, which is changed once or twice during the three hours in which they remain in alcohol; they are next wiped and transferred to chloroform, where they remain three hours, and are then put into paraffin of a melting point of 52° C., where they remain 3—4 hours. It is essential that they be passed through two lots of paraffin, otherwise they will be too soft to cut satisfactorily. The sections are examined either immediately after removing the paraffin from them, or, better still, after they have been fixed to the slide by capillary attraction. In the former case they shrink on removing the paraffin, but in the latter they expand. If fixed they must be flattened, and the surplus water removed from the slide as quickly as possible, as it abstracts colour from the section, and they must be thoroughly dried before the paraffin is washed out. If these instructions are carried out they keep well. Sections prepared in the above manner may subsequently be stained (on the slide) by Beneke's method for showing neuroglia structure with good results.

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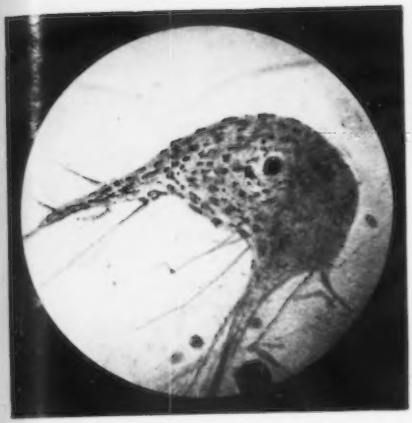

FIG. 9.

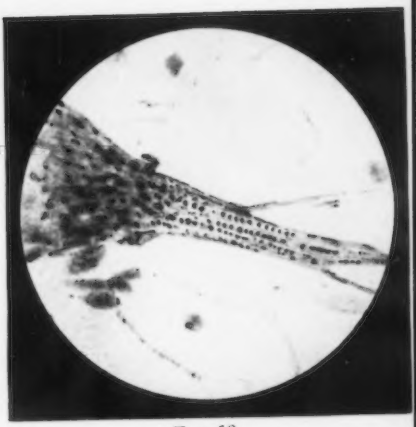

FIG. 10.

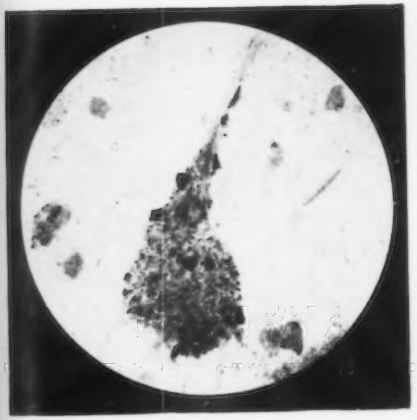

FIG. 11.

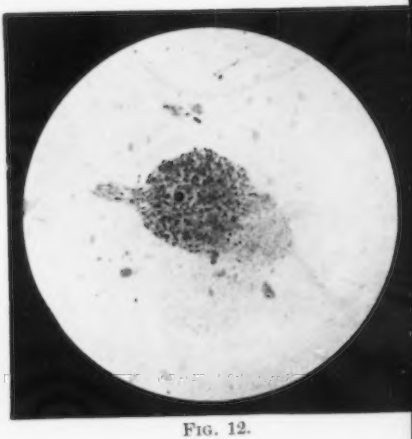

FIG. 12.

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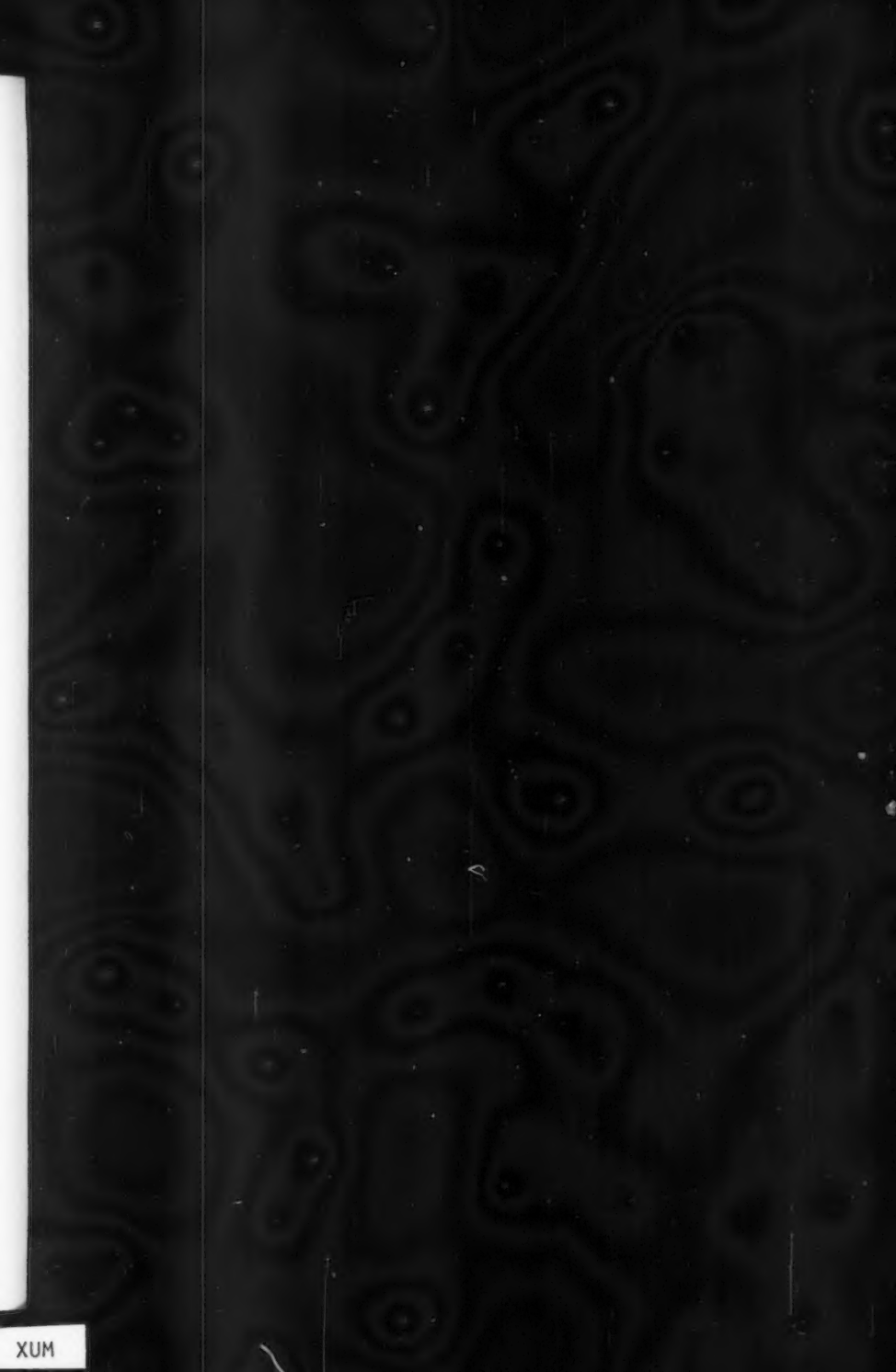
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in senile and otherwise degenerated cells to nearly a half. This is probably due to the dehydration of the section, the supposition being that in the latter the cytoplasm is largely occupied by some watery matter which the alcohol removes. A variation is indeed noted in the size of fresh cells, between those of the young and recent cases and the old and degenerated, as the following table shows:—

No.	Sex.	Age.	Diameter in μ of Purkinje's cells (mean of 10)		Form of Mental Disorder.	Duration.
			Long.	Trans.		
1	F.	64	37	30	Senile mania	Many years
2	F.	69	41	31	Senile mania	Doubtful
3	F.	33	42	32	Organic dementia	Over a year
4	M.	40	42	35	Dementia general paralysis	Some years
5	M.	45	47	33	General paralysis	Within a year
6	M.	39	54	30	Advanced general paralysis	Some years
7	M.	63	53	31	Chronic mania	Many years
8	F.	47	47	36	Chronic mania	Many years
9	M.	45	50	40	Chronic mania	Many years
10	F.	56	50	40	Chronic melancholia ...	Two years
11	F.	20	51	38	Acute mania	Recent
12	F.	30	52	43	Advanced general paralysis	Over a year
13	F.	33	56	45	Acute mania	A few weeks

But, in all cases, the latter in contradistinction to hardened cells retain their plump form and never appear shrunken. The processes also which in degenerated cells after hardening appear attenuated and darkly stained, do not in the fresh state, beyond the taking up of more colour, show any marked departure from a normal condition as regards calibre.

This shrinking of cells with alcohol, &c., in certain abnormal conditions produces vacuoles, which are never met with in a fresh state either in the cytoplasm or nucleus, and are with great probability artificially produced by dehydration. Nevertheless, they are a valuable sign of a pathological state of the cell in which its protoplasm is altered so that it contains an excess of watery matter.

No. 3 in the table was an instructive case as regards the formation of vacuoles. The cerebellum was very firm and with fresh films few Purkinje cells were to be seen.

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Hardened preparations showed a great increase and prominence of the radial fibres and a layer of large connective tissue elements in the region of the Purkinje cells giving off fibres running straight upwards in the cortex; only a few Purkinje cells remained and these were small, surrounded by leucocytes and often contained a large round or oval vacuole (13-22μ in diameter). None were seen in the fresh cells.

The study of the intimate structure of the cytoplasm revealed by Nissl's process is of the greatest importance in the pathology of the nerve cell. It already allows us to observe changes which were formerly quite beyond our ken, and no doubt it will in the future, when the more exact nature of the chromophilic material is known, be a valuable aid in our efforts to understand the meaning of these changes. Its importance in the pathology of the nervous system can hardly be over-estimated.

FIGURES.

FIG. 1.

Frontal cell from a woman, age 33 ($\frac{1}{12}$ oil, with ocular). Cell $51 \times 39\mu$; nucleolus 5×5. No Pigment. $\frac{500}{1}$.

FIG. 2.

Occipital cell from a woman, age 42, acute mel. ($\frac{1}{6}$, with ocular). Cell 40μ wide; nucleolus $4 \times 4\mu$; nucleus darker than cytoplasm and indistinct. $\frac{350}{1}$.

FIG. 3.

Occipital cell, man, aged 76 ($\frac{1}{6}$, with ocular). Lower half occupied by pigment; chromophilic threads breaking up into segments in apex. $\frac{350}{1}$.

FIG. 4.

Occipital cell, woman, age 20, acute mania ($\frac{1}{12}$ oil, with ocular). Cell 24μ wide; chromophilic matter segmented. No pigment. $\frac{500}{1}$.

FIG. 5.

Two giant cells, woman, age 33 ($\frac{1}{2}$ in., with ocular). Upper one $120 \times 75\mu$; nucleolus 12μ; clump of pigment at lower end; lower one 210×72; nucleolus 9μ. Pigment at right end. $\frac{150}{1}$.

FIG. 6.

Giant cell, woman, age 30, acute mania ($\frac{1}{6}$, no ocular); $186 \times 84\mu$; nucleolus $14 \times 12\mu$. Mass of pigment occupies and distends left end. $\frac{200}{1}$.

FIG. 7.

Giant cell, same woman ($\frac{1}{12}$ oil, no ocular). $\frac{222}{1}$.

FIG. 8.

Occipital cells with pale cytoplasm, no chromophilic matter and well-defined dark nuclei; man, age 63. Chronic mania.

FIG. 9.

Upper cell of fig. 5 more highly magnified.

FIG. 10.

Apex of cell in fig. 6, showing segmentation of chromophilic threads.

FIG. 11.

Motor cell, man, age 47. G.P.; large masses of chromophilic material at base and apex of cell; threads still seen in apex.

FIG. 12.

Senile cell, woman, distended with pigment; chromophilic matter exists as small, sparsely scattered granules, apex broken off.

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A STUDY OF A CASE OF ACUTE HÆMORRHAGIC (NON-SUPPURATIVE) ENCEPHALITIS.[1]

BY ALFRED WIENER, M.D.

Adjunct Professor of Nervous and Mental Diseases in the New York Polyclinic.

(*From the Clinic of Prof. B. Sachs.*)

THE occurrence of two distinct attacks of this disease in the same individual, with a complete recovery after the first attack, and with a quickly fatal result after the second, has induced me to put on record my observations in this case, including a report of the microscopic examination of the brain and the membranes. The study of this form of encephalitis is still in its infancy. There is no doubt that many of the cases which heretofore have been diagnosticated as meningitis, were cases of encephalitis. There is a great similarity in the clinical symptoms of these two diseases. In a recent monograph upon this subject, Oppenheim admits that the diagnosis of encephalitis can seldom be established with any certainty, and goes so far as to say, that a diagnosis of this disease is at best only a probable one. It was formerly believed that when this disease was present a fatal issue was inevitable, and that if the patient recovered, he in all probability had not suffered from encephalitis. The cases published by Strümpell-Leichtenstern, Fürbringer and Oppenheim, have shown the fallacy of this view.

In contra-distinction to the polio-encephalitis of Wernicke, which is due to multiple punctiform hæmorrhages in the region of the nuclei of the eye muscles, in the walls of the third ventricle, in the central grey matter of the floor of the fourth ventricle, and in the aqueduct of Sylvius,

[1] Read before the American Neurological Association, at Washington, May 5, 1897.

there is the form described by Strümpell in 1890, and afterwards by Leichtenstern and Fürbinger. This form is due to an acute primary inflammation of the brain, showing on *post-mortem* examination, a hyperæmic condition, with small capillary hæmorrhages scattered here and there in the hemispheres, centrum ovale, in the basal ganglia, &c.

The clinical symptoms of these two types will differ, in consequence of the peculiar localisation of the lesion in each, viz., either in the hemispheres or in the brain axis. Again their etiology is likewise the same, both forms occurring as they often do, after infectious diseases, ulcerative endocarditis, chronic alcoholism and other toxic infections. It is my impression that they are after all only different forms of the same disease. Cases have been reported in which a combination of the symptoms of both forms exist, which certainly very materially strengthens this view. The case of Freyhan published in the *Deutsche Med. Wochenschrift*, 1895, No. 34, is a particularly good example.

Infectious diseases seem to play a very important role in the etiology. Its occurrence during influenza epidemics, as Leichtenstern, Königsdorf, Schmidt and myself (two cases) have lately observed, and the case of Dr. Putnam's after mumps should keep us on the alert, and teach us to consider the possibility of its presence, and thus prevent confusion between it and meningitis. Influenza bacilli have been found in a case reported by Pfuhl, and in one reported by Nauwerck.

The history of the case which I wish to report in this paper is as follows:—

J. J. was two years and nine months of age when he was brought for the first time to the clinic of Dr. B. Sachs. This was in the month of August, 1893. The history given at that time by the mother of the little boy, was that he had fallen down a cellar stairway, striking on the stone steps with his head. When he was picked up he did not appear to be any the worse off for his fall. This happened three weeks previous to the time that he was first brought to the clinic. No visible injury was apparent at the time. On the same day of the accident, the

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child played with his brother and sister, and nothing was noticed in its behaviour to excite any alarm. On the following day the child began to become fretful and peevish. On the third day he began to exhibit a tendency to destroy things in general. He tore up his handkerchief and even the dress that he wore. On the fourth day a difficulty in his speech was noticed for the first time. The child also began to totter and fall. On the fifth day speech was much worse and the child could no longer walk; he was becoming more and more drowsy and cried very much. On the evening of the sixth day a convulsion took place. The child was at times in a constant state of restlessness alternating with stupor and screaming attacks. Then again it would have distinctly lucid intervals. Such was the history given by the mother, and on examination the following important points were brought out. Father and mother are both living, apparently healthy although somewhat ill-nourished; father is a shoemaker, mother has had chorea when a young girl, otherwise the family history in regard to any neurotic taint is negative. The family consisted when all were living of seven children, four boys and three girls. One boy died of scarlet fever with convulsions, oldest girl has had chorea. There have been no miscarriages. J. was the fifth child, born at term and with no difficulty at the labour. Learned to talk and walk at the usual time, and was perfectly healthy and well up to the time when he met with this fall. On examination we found a well nourished child, unable to walk or talk and in a constant state of restless activity, tossing itself first to one side and then to the other, moving its head forward and backward, crying very much and drooling constantly at the mouth. Child would make no attempt to stand or walk; when put on its feet, and an effort was made to compel it to walk, it did so in the form of jumps from one spot to another, showing that it still possessed power to move its limbs, but only in a very awkward and atactic manner. Patellar reflexes were present and exaggerated on both sides. Superficial reflexes were also exaggerated. Sensation was normal over entire body. Left also appeared somewhat more affected than right side. Child paid no attention when spoken to. When a strong light was held before its eyes, it made no attempt to follow it. However when the child was watched it was noticed that the eyes could be moved in all directions. Pupils responded to light and accommodation. No paralysis or paresis of the cranial nerves was observed. Urine was free from albumen and sugar. Pulse varied between 60, 80, and 120; temperature was of intermittent

type. The child was seen and examined on several occasions after the first examination, but no change was noticed in its condition until December of the same year when a gradual improvement began to set in. In February of the following year "1894," the child began to walk again. Speech showed no signs of returning until March. The child first began to utter words of few syllables; gradually its vocabulary increased, sentences were formed again, so that by the end of six months the child was almost completely restored to its former good health. I could detect no trace of its former illness. . . . I took the pains to examine the child on several occasions and always with the same result.

The child remained perfectly well until the spring of 1896, when it suffered from an attack of scarlet fever and diphtheria. I did not have occasion to see it during this illness, but I understand that the child made a good recovery. From September 15 to December 9, the child attended school; its reports from school were of the best, and the child seemed to take great pleasure in being allowed to attend. On December 9, the child complained of feeling tired, had pain in its head and back, besides this there were present catarrhal symptoms in its nose and throat. It appeared drowsy, and contrary to its former delight in being able to attend school, it was now inclined rather to stay at home and lie down; it soon became fidgety and restless again, running aimlessly about and at the same time apparently anxious and frightened. Speech disturbances again appeared. For ten days it continued in this manner, growing worse each day, until finally the mother recognizing that the child was again drifting into a state which closely resembled the first attack, brought it once more to our clinic for treatment. The child was sent home, a thorough anti-phlogistic course of treatment was advised and carried out, but was of no avail. On December 23, the fourteenth day of the illness, the child had a severe convulsion, then followed attacks of delirium, alternating with stupor and lucid intervals. There was again the constant drooling of the mouth, the child had the habit of incessantly protruding and retracting its tongue. Twitchings of the various muscles in the arms and legs were frequently noticed, probably a little more on the left side than on the right. It would make attempts to get out of bed, and had to be restrained; the head was again being tossed in all directions. On the 24th another severe general convulsion took place. On the 26th several convulsions occurred, and the stupor which followed was now more complete. Pupils on

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examination were unequal, left smaller than right, and no response to light. On the 27th there was delirium, stupor, coma, convulsions and finally death. Temperature during all this time was again irregular, and the pulse was constantly rapid. No proper examination of the optic discs could be made.

Autopsy.—This was made ten hours after death. I found the abdominal organs in perfect condition. Thorax normal, excepting slightly œdematous lungs, and a congested appearance of the upper respiratory tract. The heart was normal; the skull was symmetrical; the diploe was thick and hyperæmic over the left and right parieto-occipital regions. Inner surface of skull was perfectly smooth: the superior longitudinal sinus contained a small parietal thrombus. The dura mater appeared normal to macroscopic appearances; its inner surface was smooth and shiny, nowhere was it thickened. The pia could be stripped off in patches from the cortex. At the bottom of the sulci some difficulty was experienced in detaching it, and often pieces of cortex would come away with it. The lateral ventricles contained only a very small amount of a reddish serous-looking fluid. The choroid plexus was filled with blood; the ependyma was smooth; the third and fourth ventricles and the cerebellum were of normal macroscopic appearances. Several cuts were made through the brain substance, showing it to be markedly hyperæmic and in some places soft and œdematous. The whole brain was placed in Formalin to harden and then prepared for further examination.

Microscopic Examination.—Figs. 4, 6. The blood vessels in the pia were markedly congested and filled with blood. Here and there were small hæmorrhagic infiltrations. There was also noticed an abundant production of cells somewhat resembling the cells which normally coat the surface of the pia. Besides this, a reticulum of very fine fibres covering the entire surface of the pia, which appeared to be fibrinous. This was the appearance of the pia when stripped from the cortex. In some places on cross section, I found it closely adherent to the cortex, and presenting the same pathological picture above described. Pieces of cortex taken from the hemispheres, and sections made from the basal ganglia, pons, and medulla, all show a very characteristic picture. There were many dilated and overfilled blood-vessels (fig. 5). The walls of the blood-vessel, both veins and capillaries, are infiltrated with round cells. The intima does not appear to be affected. The media and adventia coats seem to bear the entire burden. Along the walls of the blood-vessels, and in the perivascular spaces, numerous wandering leucocytes

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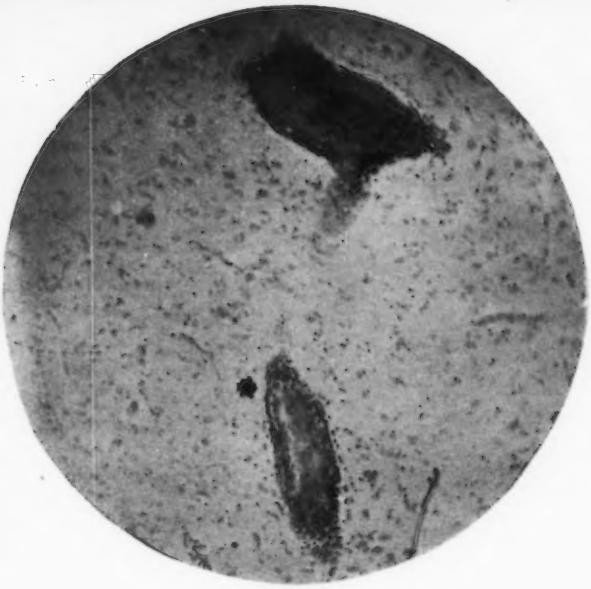

FIG. 1.

Blood-vessels filled with blood and their walls infiltrated with round cells.

FIG. 2.

Small haemorrhage with round cell infiltration. Sub-cortical.

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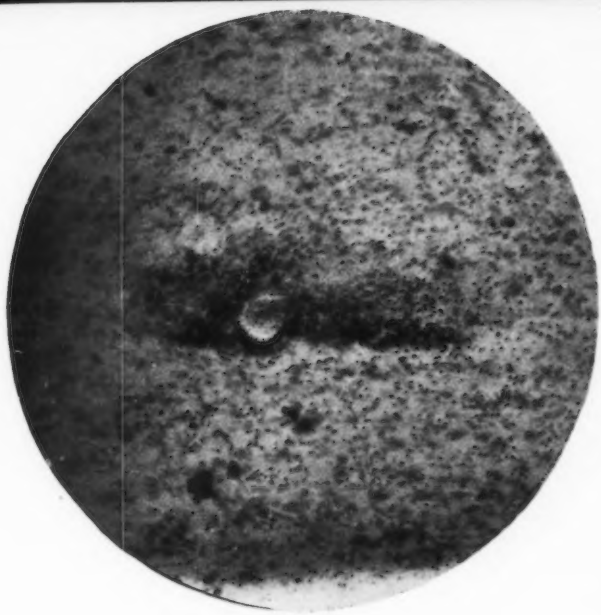

FIG. 3.
Ruptured blood-vessel. Hæmorrhage. Round cell infiltration.

FIG. 4.
Pia as stripped from the cortex. Fibrinous mass covers the entire surface.

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can be seen, together with red blood cells (figs. 1, 3). In the region of the third left frontal convolution (fig. 2), in the anterior extremity of the first temporal, and in the olivary body in the region of the twelfth nerve, capillary hæmorrhages could be distinctly seen. No necrosis of tissue, nor actual destruction of nerve elements was observed anywhere. In regard to the ganglion cells which were examined after the method of Nissl, I found that those which were situated in the neighbourhood of hæmorrhages and very much diseased blood-vessels would not take on the stain very readily. Some looked swollen, and so blurred that neither the nucleus or nucleolus could be recognised. The whole appearance resembled the picture which we recognise as cloudy swelling (fig. 7). The peri-cellular spaces were distinctly enlarged. Although several sections were stained for the purpose of bacteriological examination, no positive results were obtained. Such were the pathological changes present in this case. An acute inflammatory process in the pia and in the brain substance, with a tendency toward capillary hæmorrhages. A picture which is now familiar to us and which we recognise as "Acute Hæmorrhagic Encephalitis," or as I would prefer to say in this case, "Acute Hæmorrhagic Meningo-Encephalitis."

Let us review the clinical features of this case, in the light of these pathological facts.

A perfectly healthy child, 3 years of age, meets with an accident in falling down a stone stairway. On the following day symptoms gradually begin to appear, and at the end of a week the child has suffered from a convulsion, lost its speech, and cannot walk; its mental condition is seriously affected, the child is restless and peevish, there are attacks of screaming alternating with stupor, an irregular temperature, and a pulse at one time slow and at other times rapid, but at no time irregular. There appears to be not so much loss of power in the limbs as an awkward use of the same. The constant tossing of the head from side to side, and the occasional grasping the same with its hands, suggest the probability of headache. No attention is paid to light or sounds. No paralysis of the cranial nerves was observed. Urine was free from albumen and sugar. Optic discs and pupil reflexes were normal. Patellar and superficial reflexes were exaggerated. Such an array of symptoms we were naturally inclined to believe, when we saw this child for

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the first time, to be due to a meningitis of the convexity, following trauma. The child gradually began to improve and was well again at the end of six months. In the following three years, with the exception of an attack of scarlet fever and diphtheria, the child remained perfectly well. It developed physically and mentally to the same degree as other children of the same age. Suddenly one day it began to suffer from catarrhal symptoms of the upper respiratory tract, and began to complain and present a picture resembling almost exactly its previous attack. The child now rapidly developed the same symptoms which it had suffered from in its first attack, and in three weeks the disease proved fatal.

With regard to the etiology of the first attack, it would appear that although there was nothing present on *post-mortem* examination to show the effect resulting from a trauma upon the skull, or its membranes, nevertheless this first attack and the trauma must have been very closely associated with each other. Within twenty-four hours after the accident, the first symptoms appeared. The clinical picture that followed closely resembled that of the second attack; the autopsy proved that the second attack was due to an acute hæmorrhagic encephalitis.

That the child inherited a tendency to neurotic troubles, is apparent from the presence of chorea in the mother and in one sister. Taking these facts into consideration, we need not hesitate to attribute the first attack, if not directly at least indirectly, to the injury preceding it. Cases of this kind have been reported by Mauthner, Birdsall and Dinkler. I am somewhat at a loss to account for the second attack; it occurred, however, at a time of the year when influenza was epidemic in New York. This child began to suffer with catarrhal symptoms of the upper respiratory tract, and its general condition suggested the possibility of an attack of influenza. Again this child having had one attack of encephalitis, was naturally predisposed to this disease. This child was also beginning to attend school, and was subjected to the first mental strain.

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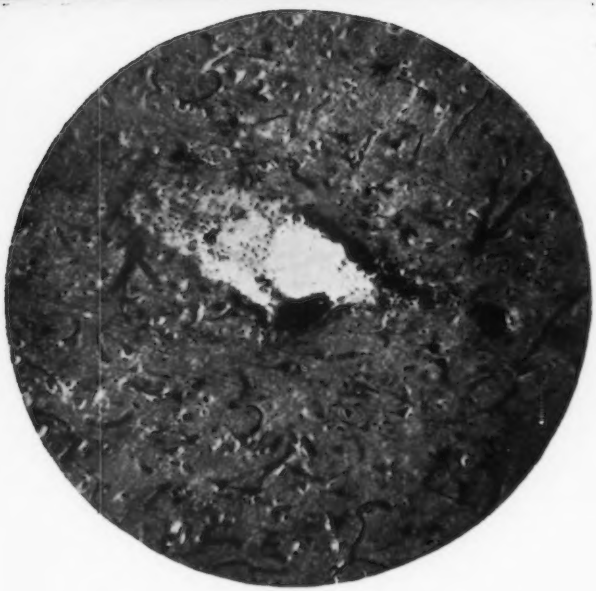

FIG. 5.

Representing a markedly hyperæmic area with a hæmorrhage in the centre.

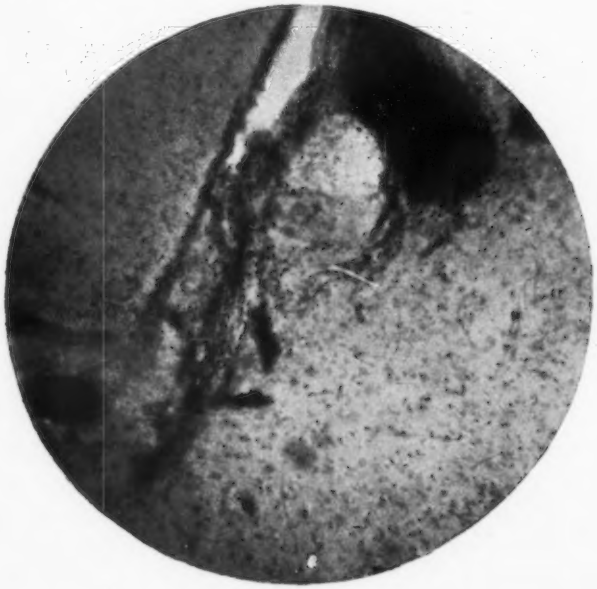

FIG. 6.

Pia on cross section at the bottom of a sulcus. Thickened and closely attached to the cortex. Infiltrated with round cells and containing

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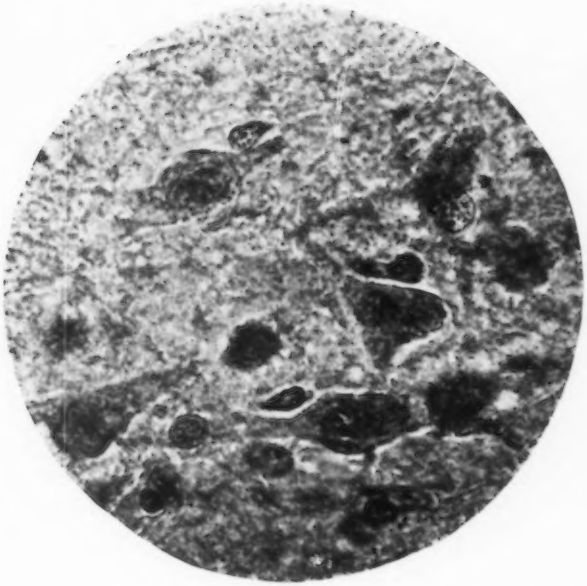

FIG. 7.

Diseased ganglion cells in the cortex, showing the cloudy swelling.

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In regard to the pathological condition found, I do not think it necessary to go into details. It thoroughly explains the clinical picture which was present. Before concluding let me direct your attention to one especial feature in this case, viz., the involvement of the pia. This has been found in the cases of Leichtenstern, Eisenlohr, and others. The condition which they found was one of œdema and infiltration of this membrane, whereas in my own case I found a picture which very closely resembles the condition found in fibrinous pleurisy. I am not prepared at present to dilate further upon this pathological fact, and wish therefore to defer further comment.

I wish to express my indebtedness to Dr. B. Sachs for the privilege of reporting my observations upon this case.

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For a complete bibliography of this subject I would refer the reader to the ninth volume of Nothnagel's "Specielle Pathologie und Therapie; Die Encephalitis und Der Hirnabscess," by Prof. Dr. H. Oppenheim, Berlin.

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FURTHER NOTES ON GRANULES.

BY ALEX HILL, M.A., M.D.

Master of Downing College, Cambridge.

In two papers published in this journal (Part LXXIII., p. 1, 1896, and Parts LXXVII. and LXXVIII., p. 125, 1897), I described a form of granule which had not hitherto been recognised in the cerebellum. These granules are carrot-shaped, the apical process, usually single, but occasionally bifurcated, tapering, bearing a few lateral branches. The nucleus lies at the thick end of the cell. A nerve fibre of great delicacy is attached to this end. The cells are of the same size as the well-known round granules which were described by Golgi, *i.e.*, 10 μ to 15 μ in diameter.

I have found these cells in the rat, kitten, puppy, and hedgehog. Judging from the closeness with which they appear in certain patches in chrome-silver preparations, they must be extremely numerous. They are not limited to the "granule layer," but are present in all three layers of the cortex of the cerebellum. I find them also in many other parts of the central nervous system.

(A) In the "granule layer" of the cerebellum they are of two kinds, as judged by the course of their axis-cylinder processes. (*a*) Granules of which the apical process is directed towards the fibres of the arbor vitæ and the axis-cylinder process towards the molecular layer. These granules are usually inclined at an acute angle to the fibres; the tapering, apical process enters the layer of fibres; the axis-cylinder process passes vertically outwards, pierces the sheet of cells of Purkinje, and bifurcates in the molecular layer in the same manner as the corresponding process of a round granule. (*b*) Carrot-shaped granules, of which the axis-cylinder process is centripetal and branched.

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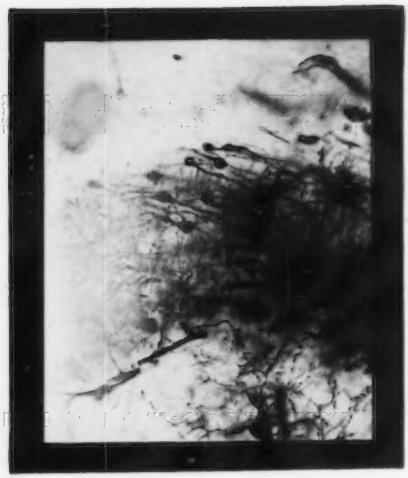

FIG. A.

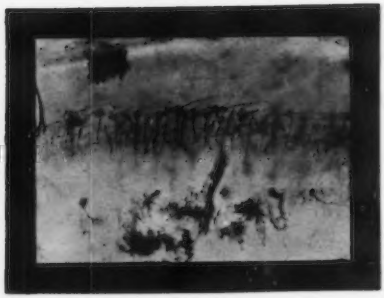

FIG. B.

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(B) In the molecular layer also, the granules are of two kinds: (*a*) Carrot-shaped granules, disposed tangentially, with axis-cylinder processes which may be traced for a considerable distance. It is interesting to notice that often the axis-cylinder processes of neighbouring granules run in opposite directions, *i.e.*, the one to the right, the other to the left. Occasionally it is seen to divide into five or six slender branches; in rare cases it loops back upon itself close to the granule. (*b*) Granules placed vertically, with their apical processes directed towards the cells of Purkinje, their axis-cylinder processes centrifugal, or *vice versâ*. In some cases in which it is centrifugal (*a*) the axis-cylinder runs vertically outwards and bifurcates; in other cases (*β*) it inclines to right or left, at an acute angle to the cell.

In an early number of BRAIN I hope to be able to give a detailed description of the various forms of carrot-shaped granule, which I have found, not only in the cerebellum, but in other parts of the central nervous system also; in the meantime the two photographs with which this preliminary notice is illustrated will remove any uncertainty as to the character of the cells, or any doubt as to their dissimilarity from every type of cell hitherto described.

Photograph A (obj. 16 mm. focal length, oc. 4, camera ·8 metre) shows a group of granules lying in the middle of the molecular layer. It will be noticed that the axis-cylinder processes of some of the granules run to the right, of others to the left. The cells of Purkinje were not stained, but they are dimly visible. A cell of Golgi appears at the bottom of the picture. The section was taken from the cerebellum of a puppy 16 days old.

Photograph B is from the same animal (obj. 16 mm., oc. 2, camera ·8 metre). The granules lie in the middle of the molecular layer. Their apical processes are directed towards the cells of Purkinje, the position of which is just recognisable; their axis-cylinder processes run almost tangentially.

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STUDIES ON THE NEUROGLIA—II.[1]

BY F. W. EURICH, M.D.

IT is a year since we roughly sketched the first results of our examination of the neuroglia by Weigert's method;[2] further investigations have now brought our studies to a stage at which a second publication appears justifiable. A certain amount of repetition of former statements will, however, be unavoidable if clearness of argument is not to suffer.

In pursuing these investigations we have had to turn aside to consider certain questions in comparative anatomy. A brief epitome of the work thus found necessary will, we think, answer the purpose of this Journal better than a detailed account.

The neuroglia, in the widest sense of the term, presents with Golgi's method three great types of cells:—(*a*) The ependyma cell, with its single branched or unbranched process. (*b*) The "astroblast," possessing almost all the characteristics of the former, but that it no longer lines the central canal; and (*c*) The fully formed "astrocyte," or "spider-cell." Two varieties of the astrocyte may easily be distinguished—the one with a small stellate body and numerous long, smooth, unbranched arms (the "fibre-cell" of Andriezen), the other with short, rough, branched processes which give it a peculiar frosted appearance. *Transitional forms between these types and subtypes can be found.* All these types may co-exist in the same animal, either temporarily or permanently; or only two may be found. But the ependyma cell is present in every instance.

[1] Being part of a thesis for the Degree of Doctor of Medicine, Edinburgh University, July, 1897.

[2] In a paper read before the Pathology Section, Annual Meeting of the British Medical Association, Carlisle, 1896.

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though it may show signs of atrophy in the adult animal; it may be the sole representative of the neuroglia, as in the amphioxus. The amount of branching displayed by the processes of the ependyma cells and astroblasts depends, *inter alia*, upon the amount of peripheral expansion which a given central nervous organ has undergone in the process of development. The presence of minute hair-like appendages on these processes appears to stand in some relation to the absence of myeline in the areas through which the processes pass; thus, in the chick the "hairs" disappear when the medullary sheaths begin to form: the same can be well seen in the spinal cord of embryonic elasmobranchs, or in the cord of the frog. Where dendritic nerve-cell processes from the grey matter are interspersed in the white columns, as in the lateral tracts of the cord of the spiny dog-fish (*Acanthias vulgaris*) or of the frog, these appendages may be retained. All the characters of the neuroglia are calculated to convey the impression that the functions of this tissue are of a passive, mechanical nature.

The spinal cord, being phylogenetically the oldest part of the central nervous system, always presents the neuroglia in a more advanced condition than do the various parts of the brain, while in the cord itself similar differences in the degree of development of this interstitial tissue may sometimes be discovered. Thus—to take one instance out of many—in the adult frog the cord contains ependyma cells and astroblasts in various stages of evolution, while the cerebral hemispheres of the same animal exhibit ependyma cells only. The higher orders and genera furnish examples to the same effect. In man, too, these differences are shown by the degree of fibrillation to which the neuroglia has attained in various parts of the brain and spinal cord. But we anticipate. For this last statement already touches on a question of histology which has been opened by the publication of Weigert's great monograph on the human neuroglia. We said "opened," but we should more correctly have said "re-opened," for Ranvier[1] had already dissented from the

[1] RANVIER, "De la neuroglie." *Arch. de Physiol. Norm. et Path.*, February, 1883.

generally accepted view (based on Golgi's silver-impregnation method) that the neuroglia consisted of a feltwork formed by the interlacing branches of innumerable cells. But the method he employed (*maceration, picro-carmine*) was too uncertain in its results. The weight of opposing authority was too great, the simplicity of the conception and the wonderful clearness and elegance of Golgi's silhouettes too convincing for his opinion to gain ground. In 1890 Weigert announced[1] that, by means of a stain which he was still trying to perfect—and which he therefore did not publish at the time—he had come to conclusions similar to Ranvier's. Ranvier's and Weigert's view was this—that in adult man the neuroglia fibres were not an integral part of the glia-cells, but were differentiated from them; that the appearance of the so-called Deiters' cells, in short, was caused by the crossing of fibres on all sides of a small mass of nucleated protoplasm, and that this illusion of a branching cell had been kept up by stains, such as nigrosin, aniline blue-black, and others, which were not sufficiently elective to show this difference between cell-body and fibre. Golgi's method was characterised as particularly liable to mislead, as it displays the cell-elements as silhouettes, and not as transparent objects, thus not permitting an insight into the true relation of fibre to cell. A few years later, Andriezen,[2] apparently without previous knowledge of Weigert's papers, published an article in which he stated that it is sometimes possible, under exceptionally favourable circumstances, to see, even in Golgi preparations, the neuroglia fibres "passing right through the cell-body." At last, in November, 1895, Weigert[3] published his monograph on the Human Neuroglia, with a description of his method of elective staining, embodying the results of many years of labour and patient study. Its appearance aroused renewed opposition, and a series of objections have been put forward, principally by v. Len-

[1] WEIGERT, "Bemerkungen über das Neuroglia-gerüst des menschl. Centralnervsyst." *Anat. Anzeiger*, 1890, p. 543.

[2] ANDRIEZEN, "The Neuroglia Elements of the Human Brain." *Brit. Med. Journal*, July 29, 1893.

[3] WEIGERT, "Beiträge zur Kenntniss der menschlichen Neuroglia," November, 1895.

hossek[1] and Kölliker,[2] which it may be worth our while briefly to examine. It is not a case of "hair-splitting," nor simply a question of the correctness or incorrectness of an unimportant point; for if Weigert's view can be proved tenable, then normal and morbid histology will tend to show that this differentiation of the fibre is, as would be supposed, a further and terminal step in the evolution and development of the neuroglia. But to proceed! Weigert's method stains neuroglia fibres and all nuclei a fine blue, leaving the protaplasm of the cell-body unstained. As for the true fibrous connective tissue, it either remains uncoloured or assumes a violet tint, according as a certain step in the process is omitted or not. The method itself in many respects resembles Weigert's well-known fibrin stain, and upon this fact v. Lenhossek's objection is based. He is of opinion that the staining reaction is due to a difference in the density of the glia-fibres from that of the cell protoplasm, and instances in support of his view, the fact that elastic fibres and other connective tissue are also stained by the process, forgetting to mention, however, that this mesoblastic tissue receives a distinctly different tint, and may even remain unstained if the action of pure chromogen on the sections be omitted. Against the argument itself we would urge the following:—(i.) That the glia-fibres are very sharply defined, so that the transition from the density of the cell-protoplasm to that of the branch (or fibre) must be an exceedingly abrupt one, a condition which, if cell and fibre (or branch) be a perfect corporate whole, would be contrary to all experience. The fact that, in the early stages of certain pathological processes with proliferation of the neuroglia (see later), the blue colour of the glia-fibre is seen to fade gradually into the pallor of the cell-protoplasm cannot militate against us; it is not a permanent feature in these cases, and *must* be noticeable at some time or other during the progress of the differentiation. (ii.) It is very hazardous to draw conclusions

[1] v. Lenhossek, "Der Feinere Bau des Nerven Systems im Lichte neurer Untersuchungen," 2nd edit., 1895, p. 186.

[2] Kölliker, "Handbuch der Gewebelehre, 1896, Bd. ii., pp. 148-150, and 791-793.

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as to the chemical or physical similarity of two bodies from similarity in their response to some one chemical or physical test; and such a test—chemical or physical—a staining reaction undoubtedly is. . . . Kölliker enters more deeply into the question. He first falls back upon the researches of Ranvier himself, who was unable to find in the cerebral cortex such differentiated glia-fibres as in the spinal cord; but if we consider that such a differentiation of the fibre from the cell probably represents the most advanced stage in the evolution of the neuroglia, and if we recall that there are phylogenetic differences in the structure of that tissue in the various parts of the central nervous system—that the central cortex, in fact, is "younger" than the cord, then we can easily understand why Ranvier failed to discover such differentiated fibres in that locality. As a matter of fact, they do occur in that region, as Weigert has shown, but they are scanty in health. Kölliker next cites Golgi; the latter had failed to obtain what Ranvier had claimed to have found, though employing the same method, and had remarked that folds in the disc-like body of the glia-cell could closely simulate the passage of a fibre over the surface of the cell body. Weigert has already answered both these objections. The first, it is clear, cannot be urged against Weigert's method, and Golgi's failure was probably due to some difference in the picrocarmine which is known to vary considerably in its staining qualities. As regards the second objection, Wiegert has pointed out that the blue fibres appear as points on transverse section, and that a fold cannot appear as a dot under the like condition. We would add that there is no reason whatever why a fold of protoplasm should assume a deep blue tint, while the rest of the protoplasm remains unstained. As a last objection, Kölliker urges that the short-armed astrocytes, with the frequent attachment of branches to vessel walls, display features which cannot possibly be explained, except by believing the fibres and the cell body to be a corporate whole. Be it so! But Kölliker must have forgotten—or it escaped him altogether—that Weigert does not claim, and never has claimed, for his method that it stains *all* neuroglia fibres; he dis-

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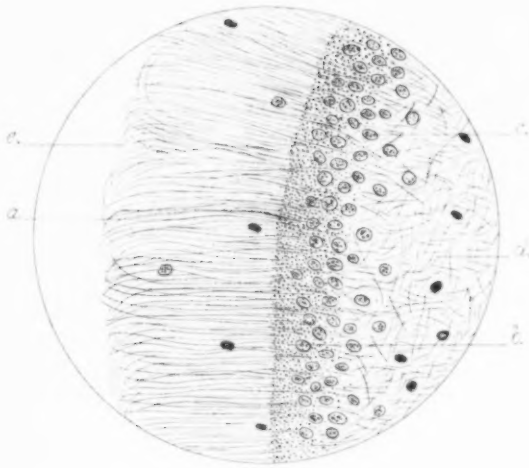

Fig. 1.

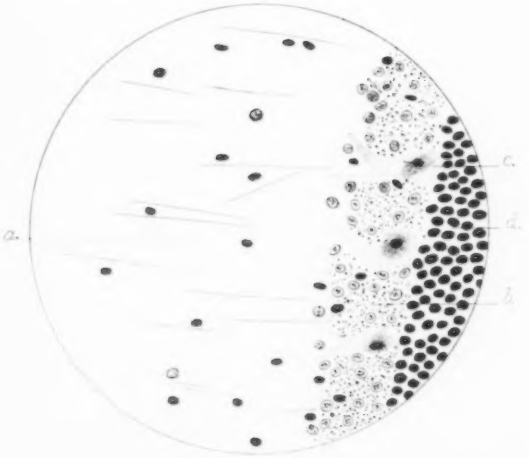

Fig. 2.

Bale & Danielsson, Lith.

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tinctly says (pp. 34 and 50, *loc. cit.*):—"there is another possibility, and it is this—that many of these cells are 'astrocytes' in the whole sense of the word, *i.e.*, that they possess not fibres but protoplasmic processes. Such protoplasmic processes, however, are not rendered visible with our method"; and again—"There cannot now remain the slightest doubt that at least the neuroglia corresponding to the so-called 'processes' of Deiters' cells (*i.e.*, the long-armed 'astrocytes') is a true inter-cellular substance, possessing, that is to say, a '*nature connective*,' in the morphological meaning of the term." Having strongly urged all these objections, Kölliker appears to have had misgivings, and propounds a theory which looks very like a compromise, and which in no way helps to simplify matters.[1] He assumes that the glia-fibres spring from a plate or disc which has become differentiated from the cell body, but still lies in contact with it.

We have seen that, from the point of view of function, the branch or fibre is the more important feature of the glia-cell. It must therefore be welcomed as a distinct advance that we now possess in Weigert's method a stain which is not only elective, but which is especially adapted for the study of the *topographical* distribution of the neuroglia.

It is not our present purpose to collect a mass of minute anatomical detail from all parts of the central nervous system. In his monograph Weigert has demonstrated that the distribution of the neuroglia in the various regions follows certain broad principles, and has himself built so extensively on this foundation, that any additions on our part would necessarily lack system. As some of these general principles are of some importance (quite apart from the correctness or incorrectness of Weigert's theory) we deem it advisable to touch upon them here:—(i.) "There is always a thick layer of closely interwoven fibres beneath the epithelium lining the ventricles and the central canal, and this is the densest feltwork to be found in the normal nervous system." (ii.) "With very few exceptions the outer surfaces present a similar concentration of the

[1] KÖLLIKER, *loc. cit.*, p. 150.

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neuroglia, but the feltwork is not so dense as in the peri-ependymal layers." (iii.) "These laws hold good not only for the inner and outer surfaces in the adult nervous system, but also for those which are present in the fœtus, and become obliterated in the process of development." (iv.) "Every medullated fibre is separated from its fellow by neuroglia-fibres." "Plexuses of glia-fibres surround the ganglian cells." Thus far Weigert! To these broad statements we should like to add that those parts of the central nervous organs which are phylogenetically the oldest, display these glia-fibres in the greatest profusion, *e.g.*, the substantia gelatinosa centralis. A good instance of this is also to be found in the cerebral cortex. We have had occasion to state that the branches of true astrocytes being protoplasmic and undifferentiated, do not stain by this method. Now, in the cerebral cortex the deeper layer of large pyramidal cells which, according to Betz, is of later development, displays practically no blue-stained fibres, though neuroglia cells can be demonstrated in it by Golgi's method. The inference is obvious; a further argument in favour of this view will be found when discussing the pathology of the neuroglia.

But if the phylogenetically oldest regions display this profusion, it does not by any means follow that every region specially well endowed with these differentiated fibres belongs to that class. All peripheral coverings of neuroglia as defined in Weigert's law iii., are examples of this; but the explanation which we venture to give of these structures will find a better place on a subsequent page. For the minute details of the distribution of the neuroglia we would refer the reader to Weigert's monograph; such details are profitless in the present instance, and we would rather turn to another branch of medical science, and note the behaviour of the neuroglia in various regions under various morbid conditions.

Anatomy and physiology owe many a debt of gratitude to pathology, especially in matters pertaining to the nervous system; for pathological processes are not events out of all harmony with nature, but obey the physiological laws of

the organism. Are there any promises from that quarter in the present instance? We believe that a wide field lies open for investigation in this direction, thanks to Weigert's method; it is a field so rich in virgin soil that we cannot do more than turn a few sods, and perhaps indicate where future workers may hope to reap a harvest. We propose, on this occasion, to confine ourselves to a consideration of *the morbid increase of the neuroglia*.

If a region be examined in which the nervous elements are undergoing a process of destruction, there will be found an increase of cell-elements, which differ in their appearance according to the method employed to demonstrate them; thus, with hæmatoxylin they appear as nuclei with a small amount of protaplasm round them; with van Gieson's stain a few processes become apparent; while, with Bevan Lewis' "fresh method" and aniline blue-black stain, they are seen to be richly branched, spider-like elements, with, may be, one, or sometimes two processes attached to the walls of a vessel. These cells have in times past been termed "neuroglial connective tissue cells," "spider cells" (Jastrowitz), "scavenger cells" (Bevan Lewis), and were considered to be mesoblastic. This conception is still upheld by Hamilton,[1] in the second part of his text-book, published only a few years ago, and finds another advocate in Bevan Lewis.[2] The mesoblastic and the "dualistic" theory will find consideration later. Every part of the central nervous system will, if diseased, present such elements at some stage of the morbid process. They are, however, most readily studied in the cerebral cortex of a general paralytic in the early stage of that disease. If such a piece of cortex be hardened, cut, and stained according to Weigert's instructions, these cells become very distinctly visible; their nuclei are blue, the protoplasm a delicate yellow (from immersion of the section in chromogen), and the processes in various shades of blue, as follows:—The distal parts of the processes are comparatively thin, and deeply stained, but as they approach the body of one of these spider-cells they broaden out, and their

[1] HAMILTON, "Text Book of Pathology," vol. ii., part 2 p. 576, *et seq.*
[2] BEVAN LEWIS, "Text Book of Mental Diseases," 1889.

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tint gradually fades, merging gently into that of the cell-protoplasm. In very advanced conditions of the disease, or in old cases of other cortical lesions, such cells are no longer seen. We find nuclei in plenty, and also quantities of blue fibrils, which pass, however, over, under, and on all sides of the nuclei, without any sign of transition into the cell-protoplasm. The fibres appear to have become differentiated from the cell-body. All stages of this process can be seen, from that in which the fibre has taken up the faintest possible blue colouration, to that in which it is difficult to say whether the fibre is still part of the cell or not. A Weigert-preparation, demonstrating the final result of such a morbid process, presents more fibres in the diseased area than are met with there under normal conditions, *but the new fibres differ in no wise from those found in health.* This process, or its final result, constitutes "sclerosis." It is clear that sclerosis is effected by the activity of the same class of elements from which the blue fibres of the normal tissue have originated, viz., the neuroglia cell. Sclerosis—*i.e.*, this formation of fibres—is, however, met with, not only in regions in which such fibres pre-exist in health, but also in areas naturally devoid of them. Such an area is the deepest layer of the cerebral cortex. While not a single blue fibre can be found there in the normal state, yet, under certain morbid conditions, a felt-work of neuroglia becomes visible with Weigert's method. Fresh sections, stained with aniline blue-black, reveal during earlier stages of disease in this layer a comparative richness in spider-cells, when contrasted with the poverty of more superficial strata. Immigration of glia-elements from the upper to the deeper layers is therefore improbable, not only for the reason given, but for another and important one, which will be referred to when discussing cerebellar sclerosis. The spider-cells, and the fibres derived from them, must have developed *in loco* from cells which had not attained to the stage of fibrillation. All these regions, of which we have instanced this cortical layer, contain glia-cells of a type other than the smooth, long-armed "astrocyte," as Golgi's method demonstrates. But if the blue-stained fibres represent the differentiated

branches of the so-called "fibre-cells," or long-armed astrocytes; and if such fibres can, under morbid conditions, be formed in regions in which only the short-armed type prevails, and if immigration can be excluded, then it follows that the short-armed and the long-armed astrocytes belong to the same class of elements, and that Andriezen is wrong when he states that the fibre-cell is true neuroglia (*i.e.*, epiblastic), but that the small bushy variety is of mesoblastic origin. We can therefore affirm that, to whatever stage of development the glia-elements of any region may have advanced, a sclerosis similar in every essential to the process described above may follow the destruction of nerve-elements.

Weigert's stain definitely confirms the glial nature of all sclerotic and cicatricial formations within the central nervous system. We have applied it to the cerebral cortex in epilepsy and general paralysis, to softenings in the basal ganglia, to sclerotic conditions of the cerebellum, to various degenerative diseases of the spinal cord and medulla, to atrophic conditions of the optic nerve—in every instance have we found the identical process, and not a sign of the proliferation of any other tissue.

Thus far it has been shown that sclerosis is in all regions fundamentally the same. We have now to add that certain variations occur, depending upon anatomical differences in the parts affected.

These variations are moreover instructive, for they allow us to recognise that sclerosis is not a confused haphazard process, but that it follows certain laws. In our former paper we instanced the spinal cord in tabes or lateral sclerosis; we showed that, in transverse sections of the white matter, the neuroglia fibres are also cut transversely, appearing as blue dots between the nerve tubules or their remains—in other words, that neuroglia fibre and nerve fibre run parallel to each other. A somewhat less prominent feature is that the septa and trabeculæ are thickened, and that the peri-vascular neuroglia is denser and more plentiful than normal. The sub-pial layer of neuroglia of which these trabeculæ are prolongations, also appears to become more

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bulky in cases of long standing—even if a healthy tract intervenes, as Lenhossek had already noted. Thus, in degeneration and sclerosis of the crossed pyramidal tract the corresponding stretch of subpial neuroglia may thicken together with its trabeculæ, while the nerve fibres of the direct cerebellar tract remain apparently healthy. That overgrowth of the neuroglial tissue does not at first seem to destroy the nerve fibres, we find also mentioned by Hamilton (*loc. cit.*) in reference to cases of "hypertrophy" of one cerebral hemisphere due to such overgrowth.

The two directions, the horizontal and the vertical, thus taken by the newly formed fibres, are identical with those followed by the healthy neuroglia; but while the horizontal disposition of the fibres is, perhaps, more favoured in the normal condition, the development of longitudinal fibres distinctly predominates in disease. What has been said of the columns of the spinal cord holds good wherever parallel bundles of nerve fibres degenerate; the optic nerves, where this condition is perhaps found in its simplest form, are an instance in point. How can these facts be accounted for? The best explanation we can think of is the following: The proliferated neuroglia cells upon which the reparative process depends tend at first to conform in shape, and in the direction of their branches, to an earlier type—a type reflecting the character of the "astroblast" in its strong main process stretching to the actual or virtual periphery, and attaching itself there. Other branches develop and increase, and take a longitudinal direction mainly. Why? The duty of the newly formed tissue, is to fill as far as possible the gaps left by the degenerated nerve fibres. This gap extends, in the case of the columns of the cord, both upwards and outwards—roughly in the shape of a cylinder, we will say. Now, any transverse axis of this cylinder is capable of being shortened by shrinking, and by gradual approximation of neighbouring tissues; but the longitudinal axis cannot count upon this help, for the longitudinal axis of any segment of the cord cannot shorten. Here the neuroglia must bear the whole burden and responsibility. That shrinkage of the transverse axis actually occurs and is an important factor.

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can be readily seen in the spinal cord from a case of tabes in which the alteration in shape is often very noticeable on transverse section, while distortion in the long axis, *i.e.*, shortening of the posterior columns, is unknown. The course taken by the newly-formed fibres in a sclerosed area appears to us, therefore, to be determined by the following principles:—(i.) A temporary reversion to an earlier type of neuroglia element at first takes place. (ii.) The processes (fibres) of these proliferated glia-elements follow the same directions as the processes of the parent cells. (iii.) The newly-formed fibres will predominate in that direction in which approximation of the neighbouring tissues and shrinkage can give least assistance in filling the gap left by degenerated nervous structures.

We have taken the tracts of the spinal cord to illustrate these principles, and have incidentally mentioned the optic nerves, but any region of sufficiently simple structure could serve as an example. In sclerosis of the cerebral cortex, for example, we find the layer of subpial tangential fibres thickened, similarly the glia-fibres around and along the blood-vessels, while the general direction of the rest of the new fibres is straight inwards towards the deeper layers. A good example is also furnished by the cerebellar cortex.

But the pathology of the neuroglia in the cerebellar cortex is interesting also in other ways. In the outermost, or molecular layer of this cortex, the neuroglia is normally represented by somewhat sparsely scattered blue-stained fibres passing radially outwards to the pia. The cells from which they have sprung lie at the level of Purkinje's cells, as Golgi's method shows. A lesion of this molecular layer gives rise to a great increase in the number of these radial fibres, which are crowded closer together, and appear a little thicker—in perfect accord with principle ii., just laid down. But this is not all. Even if the lesion of the molecular layer be a superficial one, still the whole thickness of the layer is penetrated by the newly-formed fibres—proof evident that the process of repair is undertaken by neuroglia and not by invading mesoblastic elements. And it demonstrates more than this. It shows that an increase of neuroglia over a

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certain area does not of necessity imply a primary nerve-lesion of equal extent. The thickening of the trabeculæ passing through the direct cerebellar tract in some cases of lateral sclerosis, described by Lenhossek, and mentioned above, is a further instance of this, though not quite such a clear one, as the thickness of these trabeculæ and of the subpial stratum varies within somewhat wide limits, even in health. The increase in the number of the radial fibres is, of course, accompanied by an increase of the glia-elements. But to whatever depth the molecular layer may have been affected by the lesion, these glia-elements never leave their original site at the level of Purkinje's cells. They never immigrate into the diseased molecular layer. This can be most clearly seen in sections stained with hæmatoxylin; in them a distinct line of nuclei can be discerned at or about the level of Purkinje's cells, and practically none in the molecular layer itself. That these nuclei are neuroglial in nature is evident from their presence in cases of complete destruction of all nerve-elements in that region.

On a previous page we had occasion to refer to what we considered to be a similar behaviour on the part of the neuroglia elements in the deeper layers of the cerebral cortex. If two instances suffice to make a rule—as someone has said—then the following may be formulated:—That sclerosis following a given primary nerve lesion is due to the activity of those neuroglia cells from which the pre-existing normal glia fibres (or branches) of the affected area had originated, and *that such proliferated cells do not migrate* from the site on which they were generated.

In very severe cases of cortical cerebellar sclerosis, with complete destruction of the cortical nerve elements, a few glia-fibres can also be seen, possessing somewhat of a horizontal direction, parallel to the surface, and lying, approximately, midway between the periphery and the row of neuroglia cells mentioned above. The origin of these fibres is not quite clear. We believe that they are, in fact, branches of these very cells, which branches have deviated from their course, as they spring from the cells in a somewhat bush-like fashion, and pass in diverging bundles to the

periphery. But certainly not all have this origin; some probably spring from cells in the molecular layer—cells whose nuclei were mentioned as being visible in *very* small numbers, with the help of hæmatoxylin, &c., embedded in the molecular layer. Ramon y Cajal,[1] in a recent communication, refers to certain cells in this layer which he is inclined to view as glial in nature, though they have not as yet been seen with Golgi's method. Whatever the true explanation may be, their presence does not in any way interfere with the exposition given on the preceding page.

Another form of cerebellar sclerosis is one in which there has occurred a degeneration of the more superficial cells of the *granular* layer among which the bodies of the cells of Purkinje are embedded. When a cerebellar convolution so diseased is cut transversely the neurolgia fibres in the degenerated area appear as fine blue dots, and are strictly limited to the affected part. These fibres are developed from cells lying in the diseased area itself. It will be remembered that the radial fibres of the molecular layer likewise spring from cells situated at about this level; but the two kinds of fibres do not spring from identical cells, as can be seen on careful examination, those for the radial fibres being placed a little further outwards. A consideration of the principle that newly-formed neuroglia elements resemble their progenitors in shape and distribution would also lead us to expect this.

From a study of these varieties of spinal and cerebellar sclerosis, it appears that the neuroglia proliferates and develops along definite lines; and one is even tempted to describe distinct "systems"—(such as the radial system, and another deeper one at right angles to it, in the cerebellar cortex)—were it not that that term has of late been somewhat in disrepute.

Returning to the principle of the non-migration of glia-elements, there may be added the fact that if a lesion be of such a nature as to destroy not only the nerve-elements,

[1] Ramon y Cajal, "Beziehungen der Nervenzellen zu den Neurogliazellen," *Monatsschr. für Psych.*, January, 1897. Translated from *Revista trimest. micrograf.*

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but the glia-elements also (*e.g.*, a hæmorrhage), then the area so destroyed never undergoes complete cicatrization, but always exhibits a gap or cyst.

But it may be asked—assuming this non-migration of the cells, and granting that the newly-formed cells revert at first to an earlier type—"Have the neuroglia cells of the embryo, then, no power of locomotion, and if so, how do they come to occupy peripheral positions?" We believe that they do not migrate, even in the embryo; that they possess no power of amœboid movement as some authors appear to suppose. One causative factor in the change of position may be their rapid multiplication, by which they are crowded outwards. The chief mechanism, however, appears to us to be the rapid growth of the nervous matter itself. Our theory of the process is this:—The distance between the central canal and the periphery at which the branches of the astroblasts terminate is gradually increased, and unless these branches lengthen the cell body must be drawn outwards. While being drawn outwards they multiply. The cells thus generated being nearer the periphery have a relatively shorter main process, the bifurcation of which takes place nearer the cell body than in the old position. In the further outward passage of succeeding generations this point of bifurcation approaches the cell still more—as Golgi's preparations of suitable material clearly demonstrate. This growth of the nervous centres while increasing the area, has of course also increased and expanded the circumference; the distance between the points of attachment of the divisions of any neuroglia process must, therefore, be increased and their cell of origin drawn up to the periphery; the branching divisions of the process thus gradually apply themselves to the periphery and get to lie parallel to the pia mater. We would give this in explanation, not only of the change in position of the astroblasts and glia-cells in general, but also of the formation of the subpial feltwork, and its prolongations along the vessels. The fact that this subpial layer of neuroglia is present only in the higher classes of animals, and that it is densest in those regions which are, phylogenetically, the oldest, may also in this way be explicable.

Lastly, our theory will—if correct—tend to show that the arrangement and disposition of the neuroglia cells are to a large extent directly brought about by the growth and development of the nerve-elements themselves. In other words, the insulating and supporting structures are procured and arranged mechanically by the growth of those very nervous organs which are to be insulated and supported by them.

One question still remains to be discussed, a question which has already once or twice crossed our path. Is the neuroglia purely epiblastic? Or does it contain elements from the mesoblast also? Or is it altogether mesoblastic in origin? The last can, we think, be summarily dismissed. Epiblastic elements certainly form the neuroglia of the embryo, and the point at issue is this:—Are the cells as found in the adult all derived from these embryonic glia-elements? If we believe that each individual neuroglia cell is directly derived from an ependyma cell, and has to go through every stage of development, from that of the astroblast upwards, then indeed the difficulty is great; for not only does the formation of astroblasts appear to cease long before the nervous organs have attained their full development, but we must also mentally endow the glia-cells with the power of spontaneous movement. Refuge has usually been taken[1] in supposing that undifferentiated spongioblasts have moved outwards in the process of development, and give birth to astrocytes without passing through the astroblast stage. But neuroglia-cells can multiply to form a sclerosis in the adult nervous system, and why cannot they also divide in the embryo? Or are we to assume that a sclerosis, too, is brought about through the agency of such undifferentiated spongioblasts? The whole mechanism of the sclerotic process, as described in these pages, is contradictory to this view. If, however, we adopt the theory with which we have attempted to explain the position of the astroblasts and their derivatives, then these difficulties are to a large extent avoided. We must, on this theory, believe

[1] *E.g.*, v. LENHOSSEK, "Der Feinere Bau des Nervensyst," 1895.

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that every astroblast and astrocyte is capable of multiplying, and that their offspring are characterised by possessing a process standing out from among the rest—thicker, stronger, and passing to the periphery or to a blood-vessel—this process being *the* great family feature. It has been shown in the foregoing that when the branches have become differentiated into fibres they can (in man) be stained according to Weigert's method, and that such fibres can be developed by both the long-armed and the short-armed type of astrocytes. Both these types must therefore belong to the same tissue. Transition forms can, moreover, be found in man, and especially in the lower animals. On all these grounds—both normal and pathological—we feel ourselves justified in believing that the neuroglia is a tissue of epiblastic origin without a trace of the admixture of mesoblastic elements. Positive proof can, however, only be furnished by the discovery in the embryo of mitoses in situations known to be occupied by neuroglia cells only. Investigations on this point are, however, still a desideratum.

In the foregoing pages we have more than once had occasion to express the belief that the function of the neuroglia is a passive one. It is, however, only right to state that other theories ascribing more active and complex properties to that tissue have been put forward by various authorities. Thus Ramon y Cajal, though believing in its insulating properties, is also of opinion that the glia-cells and their branches can expand and contract (like the pigment-cells in the cutis of the frog), and thus prevent or bring about the contact of different nerve-elements, especially of terminal ramifications; in this way he tries to explain associated nerve-action, inhibition of such associations, sleep, &c. The culminating point of his theory is that the will (!) acts on the neuroglia cells and determines the condition of relaxation and contraction. It is self-evident that this theory is absolutely incompatible with Weigert's views as to the histology of the human neuroglia; and it has already been dissected and severely criticised by Kölliker. We can only marvel that a man of

Cajal's mental calibre and scientific accuracy could be tempted to such a theory.

Another function is claimed for the neuroglia by Bevan Lewis—still believed in by some, especially by alienists. According to this, the "Scavenger-cell theory," the glia-cells possess not only the function of removing detritus, but also the power of attacking disease nerve-cells and fibres. What are the facts? Bevan Lewis builds his theory upon the following. Numbers of "spider-cells appear where nervous structures undergo destruction and invade the nervous tissues, which "spider-cells" are attached to vessels by one or more processes and send branches to nerve-cells which they may encircle. The "spider-cells" form fibres and degenerate when the broken-down nerve-tissue is removed. Nerve-detritus can be found in these "scavenger elements." Lastly, domestication is said to have a deteriorating effect on the mental capacities of animals, such as the sheep and the ox; and "scavenger-cells" can be seen in the cerebral cortex of these beasts. To this we would reply: firstly, some twenty years ago Weigert pointed out that any two tissues are preserved in equilibrium by mutual resistance; atrophy of the one lessens such resistance, and proliferation of the other results. Every organ in the body can show examples of this under morbid conditions, yet we would not infer that the proliferated tissue in such cases was playing the part of a scavenger. The neuroglia is no exception; atrophy of nerve-tissue is compensated by growth of neuroglia. In obliteration of the central canal—which furnishes an instructive example—the cubical cells become loosened, and packing the lumen of the canal, are followed by an increase and invasion of neuroglia, fibres from the latter passing between them in all directions. Yet these cubical cells are not attacked by the spider-cells which precede the stage of fibrillation. Nerve-cells, too, may die and yet not be removed; they may calcify instead, as in the hippocampal gyrus in epilepsy; secondly, the attachment to vessels and the feltwork round ganglion cells are normal histological conditions; the latter appearance cannot, there-

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fore, be looked upon as evidence that the cell is "attacked" by the "scavenger elements" The increase in this vital activity makes the latter more susceptible to stains, such as aniline blue-black. Further, no proof has ever been furnished that the granules visible in the bodies of spider-cells are composed of nerve-detritus, and are not rather produced by a senile change in the glia-cells themselves; lastly, the glia-elements in the cortex of such animals as the sheep and ox are not in so advanced a condition as those in man; they do not represent a hypertrophied state, having never been smaller; the glia-cells in almost all parts of the nervous system in the lower mammalia are relatively larger than those in man; no sign of degeneration can be found in the cortical nerve-elements themselves. Our interpretation of these "scavenger elements" is, therefore, this, that they can but constitute a form characterising the neuroglia-cell in one period of its life history, and that in any proliferative and reparative process on the part of the neuroglia, this earlier stage must, in the natural course of things, be reverted to before fibrillation, as the final result, can be attained.

Summing-up the results of these "studies" we find that:

(i.) The ultimate stage in the development of the neuroglia cell is its separation or differentiation into free fibre and cell-body; and that this condition is found to a varying extent in different regions.

(ii.) That not all glia-cells reach this stage, but remain as true astrocytes, with protoplasmic branches.

(iii.) Every reparative and sclerotic process is the work of the neuroglia; the fundamental principles of all such processes are the same.

(iv.) These principles consist in:—(*a*) a reversion in type; (*b*) the fibres follow the paths indicated by the pre-existing branches or fibres; (*c*) newly formed glia-cells do not migrate; (*d*) the predominance of fibres in any one direction depends upon the amount of help afforded by shrinking and approximation of surfaces in the other directions; (*e*) both types of astrocytes are capable of developing a sclerosis.

(v.) The neuroglia is in all probability an epiblastic structure, without admixture of mesoblastic elements.

(vi.) The position of the glia-elements in the healthy adult is probably determined by the growth of the nerve-elements themselves.

(vii.) There is no evidence of mesoblastic connective tissue elements assisting in a sclerosis.

(viii.) In sclerosis each glia-fibre has its own area, which area is that "supplied" by the cell from which it has sprung; and each fibre keeps to its own area. In other words, there are "systems"—*sit venia verbo*—of glia-fibres, both in health and disease.

Addendum: Since the above was written, an article on this subject from the pen of Dr. W. F. Robertson has appeared in the *Journal of Mental Science* for October, 1897. This writer differs from us in that he denies a *separation* of fibre from cell, but allows a *differentiation*. To criticise his work would be out of place on the present occasion. We would only point out that, granted but such a differentiation, all the points deduced in this paper still hold good.

DESCRIPTION OF PLATE.

FIG. 1.

Sclerosis of cerebellar cortex; total destruction of nerve-tissue. (From a case of chronic epilepsy.)

a = Sclerosed molecular layer, with its longitudinal fibres.
b = Sclerosed peripheral zone of granular layer, its fibres cut across.
c = Neuroglial nuclei.
d = Sclerosed medulla.

FIG. 2.

Sclerosis of peripheral zone only of granular layer of cerebellar cortex. (From a case of general paralysis.)

a = Molecular layer; no sclerosis.
b = Sclerosed outer zone of granular layer.
c = Cell of Purkinje.
d = Apparently healthy part of granular layer.

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Critical Digests.

ON THE TRACTS OF THE SPINAL CORD AND THEIR DEGENERATIONS.

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Introduction.

It is my object in this digest to confine myself as strictly as possible within anatomico-histological limits. I propose to review at length the observations of those who, as anatomists or histologists, have investigated, experimentally or otherwise, secondary degenerations and scleroses; to consider the publications of workers on the normal anatomy, on the development, and on diseases of the spinal cord, only in so far as they bear upon the spinal nerve tracts; and in conclusion to briefly refer to the changes which occur in the spinal cord after amputations of extremities.

The importance of the contributions which have been added to our literature on secondary degenerations of the spinal cord during the past decade, and the augmentation of our knowledge accruing therefrom, cannot be over-estimated. A reason for the refreshing zeal and industry displayed by workers on this subject is readily found; its mainspring has undoubtedly been the discovery and elaboration by Marchi of his now famous osmium-bichromate method. In the same way as the introduction of the silver method of Golgi has stimulated the neuro-histologist, so the method of Marchi has given a fresh impetus to the neuro-pathologist and proved an especial boon to the experimenter on secondary degenerations; by reason of the delicacy of its working even sparsely disseminated degenerated nerve fibres can be traced with perfect ease and accuracy, and thus many breaches left open by the method of Weigert have now been satisfactorily filled.

To suit purposes of convenience, I shall adopt as my scheme in this periscope the arrangement of the various columns or tracts of the spinal cord into the two main physiological groups,

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ascending and descending, and I shall discuss the points concerning the development, the origin, course, connections, and termination of each column individually.

The degenerations which result from spinal hemisections, from sections of nerve roots, &c., and from cerebral and cerebellar lesions, will not be dealt with under special headings, but they will be referred to under the particular spinal tracts which they affect.

Pyramidal or Motor Tracts.

The pyramidal or motor tracts have certainly been more attentively studied of late years by experimental neurologists and neuro-pathologists than any other spinal tract, and consequently there is no other tract concerning the course, the connections, and the functions of which we are more enlightened. Yet, while congratulations are due to the various investigators who have assisted in bringing our knowledge of the tract to this advanced stage, there are, nevertheless, several points, as will be presently indicated, bearing more especially on its connections and its termination, which demand further elucidation and prevent us from declaring that our researches on the subject are yet complete.

Development.—In regard to development, Flechsig's most valuable researches have definitely proved that in the human fœtus there is no trace of myelinisation of the fibres of the pyramidal tract until the end of the normal period of gestation, and yet certain other white columns receive their medullary investment, and can be readily recognised on that account as early as the fifth month of intra-uterine life. Then Bechterew has shown, that in those animals which are endowed at birth with the power of locomotion, the pyramidal tracts are almost completely developed before the termination of the period of gestation. Bechterew has also demonstrated that in animals possessed of coarse movements only, such as the whale, the pyramidal tracts never attain a high stage of development; and it may be accepted as a general rule of comparative anatomy that the degree of development of these tracts is proportional to the multiplicity, complexity, and delicacy of the movements of the animal.

In a research having as its object the determination of the period which elapses before secondary degeneration becomes evident in the various tracts of the spinal cord after an experimental lesion, Schäfer arrived at the conclusion that an analogy exists between the time of onset of secondary degeneration and the period of development of the medullated sheath of the fibres composing these and other tracts. Schäfer employed the cat in

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his researches, and after a hemisection of the cord found that degeneration commenced in the column of Goll on the fourth day after the operation, it appeared in the dorso-lateral cerebellar tract on the sixth day, but it was not until the fourteenth day that it became evident in the one of tardiest development, viz., the pyramidal tract. Worotynski has written in confirmation of Schäfer's observations.

Volume.—Recent work confirms the variability in relative volume and representation of the anterior and lateral pyramidal tracts one to another in the spinal cord. According to Flechsig's original doctrine, their relative volume varies with the extent and completeness of the crossing at the point of decussation in the medulla oblongata. In 75 per cent. of cases this decussation is relatively equalised, and well developed anterior and lateral pyramids, which we may regard as normal, are to be found. In the remaining 25 per cent. the crossing is either unequal, incomplete, or excessive; if the decussation be unequal, that is to say, if there be more fibres crossing from one side than from the other, then in that side from which the major number of fibres decussate the pyramidal tracts, both anterior and lateral, will be poorly represented; if, on the other hand, the decussation be incomplete, then the relative size of the direct pyramidal tracts to the lateral ones will be increased above normal; and if, finally, the decussation be complete, then the condition is arrived at which Boyce, Sherrington, Singer, and others have proved to exist in some of the lower animals, viz., the cat, dog, and monkey, in which there is an almost complete absence of the direct pyramidal tracts and relatively large sized lateral tracts.

Length.—In the same way as the volume of these tracts is for the above-mentioned reasons exceedingly variable, so their length is never exactly equal in different individuals, a fact which accounts for a number of discrepancies which appear on reading the observations of various writers. Thus, whereas Bouchard told us that the direct pyramidal tract terminated in and did not descend lower than the mid-dorsal region, Tooth has traced it to the second lumbar segment, and the most recent observers of all, Déjérine and Thomas, assisted by the method of Marchi, have now followed it down to the level of the sixth sacral pair of nerve roots. Then in regard to the lateral pyramidal tract, Loewenthal and Vulpian fixed its termination at a point opposite the origin of the second lumbar pair of nerve roots, Tooth saw it opposite the fourth lumbar roots, and Déjérine and Thomas now find that it actually ends in the conus medullaris, but ceases to exist as a compact bundle at the level of the fourth sacral roots.

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Origin and Course.—That the trophic cells for the fibres of the pyramidal tracts are the large pyramidal or multipolar cells situated in the motor region of the cerebral cortex, *i.e.*, the ascending frontal and ascending parietal convolutions and the posterior parts of the first and second frontal gyri, is almost unanimously agreed. But a destructive lesion involving these parts gives rise to secondary degeneration, not only of the recognised motor tracts, but also of other systems of fibres; and though it is not definitely known that the performance of motor acts is dependent upon these other systems, yet it is more than probable that there is some relation, and since the view that a pure motor act is considerably more complex than it was originally supposed to be is every day gaining more supporters, it is possible that these systems aid in the production of that complexity, and it will, therefore, be necessary to mention them.

In the first place Monakow, Redlich and others, after destruction of the motor area of the cat, found marked evidence of degeneration in various parts of the thalamus opticus; the same observers, and also Meynert, likewise found degeneration in the substantia nigra of Sœmmering. Redlich in the human being, as well as in lower animals, has constantly found degeneration of fibres leading to cells in the nuclear matter of the pons, and he suggests that these are collaterals of the pyramidal fibres, and that they are connected by means of a second neuron[1] with the opposite cerebellar hemisphere. Also there have been found degenerated certain fibres of the lemniscus (viz., the lateral pontine bundle of Schlesinger) and internal and external arcuate fibres; and, lastly, fibres have been followed from the pyramidal tract to certain cranial nerve nuclei, viz., the facial and hypoglossal (Hoche).

One has only to contrast the small size of the pyramidal tracts as they lie side by side on the ventral aspect of the medulla oblongata, with the size of the tracts in the crus for instance, to be convinced that an appreciable number of fibres must diverge in the manner above indicated, and Starlinger's experiments on the dog seem to prove that these diverging fibres possess a most important bearing on motion, for he found that section of both pyramidal tracts in the medulla oblongata was attended by no lasting interference with movement, whereas the usual paralysis

[1] Throughout this digest the term "neuron" is employed in its broad and more generally accepted sense, *i.e.*, it includes a cell body along with its dendritic processes or neuro-dendrons and its axis-cylinder process or neuraxon. This note is deemed necessary because some apply the term to the axis-cylinder process only.

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followed a lesion of the motor area in the same animal. Redlich obtained like results in the cat, and to account for the opposite consequences in these two operations, they conclude that some fibres other than those embodied in the pyramidal tracts in the medulla oblongata must exist as channels for the conduction of motor impulses, and that some of the diverging fibres above-mentioned must form some of these channels. Doubtless fresh researches will shortly shed new light on this point.

Otherwise there is nothing new to remark concerning the disposition of the motor tract in the internal capsule, the pes, the pons, or the medulla oblongata above the decussation. The exact nature of the decussation of the pyramidal fibres at the lower end of the medulla and elsewhere is, however, still a source of fruitful discussion. This portion of the tract has been dealt with at length by Rothmann; and Boyce, Mott, Déjérine and Thomas, Redlich, and others have made incidental references to it in connection with remarks on the occurrence of bilateral degeneration in the spinal cord in consequence of a unilateral cortical lesion, and since these two subjects are inseparable they will be dealt with together at this stage.

(*a*) *The Pyramidal Decussation;* (*b*) *Bilateral Spinal Degeneration in consequence of a Unilateral Cerebral Lesion.*—Bilateral degeneration in consequence of a unilateral cerebral lesion was first drawn attention to by Westphal as early as 1875. Pitres worked the matter up very thoroughly some years later, Muratoff and others have obtained the degeneration in dogs, Mott and others have found it in the monkey, and Boyce and others have seen it in the cat. Numerous and intricate theories have been promulgated to explain this bilateral degeneration, and though most of these are happily now of historic import only, yet it will be interesting to give a brief outline of the more important ones.

Firstly, Charcot believed that in addition to the usual decussation in the medulla oblongata there occurred an extra crossing in the anterior commissure of the cord, but the method of Marchi overthrows this view by failing to reveal degeneration in this situation. Then Hallopeau, who received the support of Marie and Pitres, thought that at the decussation in the medulla oblongata the fibres from either side come into such close contact with one another that direct extension of the inflammatory process from the diseased to the normal fibres must occur.

Passing over the work of Franck, Moeli, and Schäfer, we next come to the very thorough experimental investigations of Sherrington, which led him to assume that the degenerated fibres,

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which are to be seen descending in the lateral pyramidal tract on the same side as the lesion, are derived from fibres of the opposite lateral tract, which, having already decussated at the usual situation, leave it to recross in the grey matter of the spinal cord and regain the side of the lesion without coming into connection with ganglion cells in the grey matter through which they pass. For these fibres he suggested the name "re-crossed pyramidal fibres." Sherrington's observations received support from Unverricht, Kusick, and Vierhuff; but, on the other hand, Loewenthal, Fürstner, and Knoblauch, who practised like experiments, did not get similar results.

Now it must be mentioned that all these observers worked before the method of Marchi, which is so exquisitely adapted for picking out isolated degenerated fibres, was discovered, and as they could only practise the staining methods of Weigert, &c., much of the value of their researches must be discounted.

Since the introduction of Marchi's method the matter has been taken up, amongst others, by Sandmeyer, Muratoff, Boyce, Mott, Hoche, and Rothmann.

Sandmeyer obtained bilateral degeneration nine days after experimental cortical lesions, but offers no explanation as to its occurrence.

Boyce and Mott in the cat and monkey respectively, Déjérine and Thomas in the human being, Muratoff in the dog and in the human subject as well, endeavour to explain the homo-lateral degeneration by a direct passage at or about the level of the decussation of fibres from one cerebral hemisphere to the lateral pyramidal tract on the same side as the lesion.

Marchi, as a result of experimental investigation, attributes the bilateral degeneration to an extra decussation higher up, viz., in the corpus callosum. This idea is supported by Hamilton, but it has been practically nullified by the work of Muratoff and others.

We lastly come to the observations of Rothmann, who examined the spinal cords of dogs operated on in Munk's laboratory and obtained the bilateral degeneration in every instance. His view as to its production is in a sense a revival of that of Hallopeau above mentioned, viz., that it originates at the pyramidal decussation, and that it is due to compression of the bundles of fibres from the healthy side by the swollen bundles of degenerated fibres which are coming from the side on which the lesion was. He urges in support of this that four weeks after the operation the homo-lateral degeneration is no longer recognisable, and publishes convincing drawings to illustrate his views.

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From the foregoing it is painfully obvious that the anatomical disposition of the pyramidal fibres at the decussation is by no means clearly understood, and that the source of the bilateral degeneration is not agreed upon.

Topography and Termination of the Lateral Pyramidal Tract.—In regard to the topographical distribution of the lateral pyramidal tract in the spinal cord there is little new, only the method of Marchi has shown that its limits are not so sharp as was formerly supposed, and that scattered aberrant fibres are to be seen in considerable abundance in the columns situated immediately ventral to the old boundary of the tract. These fibres are especially noticeable in the cervical region.

We still require enlightenment as to how and where the pyramidal fibres terminate in the grey matter. Kölliker, Golgi, Ramon y Cajal, Sherrington, and Lenhossek, mainly aided by the method of staining by metallic impregnation of the myelin, have shown that the pyramidal fibres at intervals in their course along the cord issue numbers of collaterals, and Lenhossek claims that these collaterals are not directly connected, as one would suppose, with the ganglial cells of the anterior cornua, but only indirectly so by the intervention of cells of the posterior cornua to which they course and around which they arboresce. This statement, however, requires confirmation. Then, in regard to the termination of the main trunks of these fibres, physiologically proof is not wanting that they are connected with the multipolar cells of the anterior cornua on the same side, but anatomically that link of the fibre, situated between the lateral column and the anterior cornual cell, has not yet been satisfactorily defined. Experimental workers (Starlinger, Monakow, Rothmann, Redlich) employing the method of Marchi, have been unable to trace degenerated fibres from the lateral column to the anterior cornual cells; but it is possible that these observers examined the cords of their animals too soon after the operation, and that they would have detected degeneration if they had allowed them to live longer; because in the case of the spinal cord of a man examined by the writer, in which there was profound descending degeneration in consequence of an extensive cerebral thrombosis six weeks old, the method of Marchi showed distinct darkening of the myelinic plexus in the anterior cornu on the side of the degenerated lateral pyramid, though a connection between any fibre in the lateral column and any anterior cornual cell in its entirety was not traceable. Of course, the difficulty here is that not only is the course of the fibre apparently tortuous, but also it

more or less entirely sheds its myelinic investment as soon as it enters the grey substance, and therefore cannot be clearly followed by a method which has for its object the display of disintegrating myelin.

Monakow discredits the existence of a direct connection between lateral column fibre and anterior cornual cell, and assumes that between the spino-muscular or anterior cornual cell neuron and the cortico-spinal or pyramidal cell neuron, a third neuron is interposed, composed of a cell after the nature of Golgi's second type, and that this cell is situated in the region of the processus reticularis.

Anterior or Direct Pyramidal Tract.—Turning next to the direct pyramidal tract, it has been proved that this tract practically does not exist in the cat or the dog, and likewise in the monkey there appears to be no adequate homologous representation; also, in the light of recent research, the tract is being divested of much of the importance it originally possessed in the human being, and is considered to be less extensive and less voluminous than Ludwig Türck, its original discoverer, taught us to believe. Türck's description of the tract was founded on observations of the changes in the cord resulting from capsular cerebral lesions, and not from cortical lesions. Now the difference between the degree of degeneration in the anterior pyramidal tract after a cortical lesion contrasted with that after a pontine or a capsular lesion is exceedingly marked. In the case of the latter the degeneration in the antero-internal column is profound and complete, but in a case of destruction of the cortical motor sphere the direct pyramidal tract degeneration is never more than partial, that is to say, the degenerated fibres are disseminated throughout it, and are surrounded by an equal or greater number of healthy fibres. These points of difference are well seen in a number of specimens of sections of cords of human beings prepared by the writer. There is no entirely satisfactory explanation of the difference in degree of the degeneration in the anterior pyramid in these two lesions; it can only be assumed that certain fibres which course down in the antero-internal tracts are not derived from the cerebral cortex, but take their origin from structures in the capsular region. For instance, Boyce's cerebral hemi-extirpation experiments in the cat show that the fibres of the posterior longitudinal bundles descend in the antero-internal tract on the same side as the lesion. I am not aware that these fibres have been traced so far downwards in the human being, but it is possible that they can be, and that the source of these fibres, by

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Bechterew supposed to be in the optic thalamus, is destroyed in a capsular lesion, whereas it of course escapes in a cortical one.

The anterior pyramidal fibres, like the crossed ones, issue numbers of collaterals in their downward course, and these collaterals, and likewise the main trunks of the fibres, are believed to pass over in the anterior commissure, and to terminate by arborescing round cells in the opposite anterior cornu.

"*Retrograde Degeneration*" *in the Pyramidal Tract.*—The name "retrograde degeneration" has been assigned by Sottas to an ascending change in the pyramidal tracts, which that author observed in four cases of medullary syphilis, in which the lesion was a softening followed by sclerosis, situated in and extending for a variable distance along the dorsal region of the spinal cord. The ascending change was prolonged upwards into the cervical region, it diminished in intensity as it ascended; topographically it corresponded, more or less exactly, to the distribution of the lateral pyramidal tract, and finally it disappeared in the upper part of the cord.

Gombault and Philippe have described similar instances of "retrograde degeneration" in two cases of syringomyelia and two of myelitis; in one of the cases of syringomyelia there was ascending sclerosis of the anterior pyramidal tract, as well as of the lateral.

This retrograde alteration possesses the following characteristics, which distinguish it from Wallerian degeneration. It is of tardy development; it undergoes a gradual diminution in intensity and extent as it ascends, and it has certain quite distinctive histological features. These are, that on transverse section the affected tract seems to be riddled with circular or oval lacunæ of varying dimensions; examined more closely, these lacunæ appear to be altered and enlarged nerve fibres. In the centre of some an axis cylinder is still clearly visible; often normal, sometimes swollen and not susceptible to stains. The spaces frequently contain *débris* of an indefinite nature, and the delicate rim investing them is probably the altered neurilemma or primitive sheath, the myelin of the fibre having disappeared. Throughout the affected tract healthy fibres are disseminated; these increase in number as one ascends, but in the immediate neighbourhood of the lesion the lacunæ greatly preponderate.

The method of Marchi only shows scattered fibres undergoing degeneration. This retrograde change might receive divers interpretations, but, as Gudden, Forel, Monakow, and Darkschewitch have shown, when a system of fibres becomes separated

from its trophic centre, it, as a matter of fact, degenerates in two directions, centripetally as well as centrifugally, but the centripetal degeneration is the least pronounced, is best observed in young animals, and only evidences itself after a more or less prolonged period. The latter condition was fulfilled in all Sottas' and Gombault and Philippe's cases.

It would appear that the changes in the spinal cord in "retrograde degeneration" bear some resemblance in a pathological sense to those seen in the cord in certain cases of amyotrophic lateral sclerosis, viz., those cases in which the disease first definitely proclaims itself in the spino-muscular neuron, and thence spreads upwards along the cortico-spinal neuron, and not those cases such as one described by Mott, in which the degeneration seemed to appear more or less simultaneously along the entire motor neuron. To explain the degeneration of the motor neuron in amyotrophic lateral sclerosis, neuro-pathologists, accepting the neuron theory as their basis, assume that its initial cause is some obscure loss or diminution of function in the trophic cell of the neuron, situated in the motor cerebral cortex or the anterior cornu of the spinal cord. Now, it is readily conceivable that retrograde sclerosis can be explained on the same theory, for, given an initial destructive lesion in the course of any neuron, we, in the first place, have a more or less immediate necrosis of the distal portion of that neuron; then, secondly, since there is no further call for the service of the central nerve-cell, either in its motor or its trophic function, it can reasonably be assumed that that cell, and likewise the portion of the neuron situated central to the lesion, must naturally undergo a slow process of atrophy, which will ultimately lead to total decay.

If the above view be correct, then it is evident that "retrograde degeneration" cannot possibly be confined to the pyramidal tracts, but must affect other systems of fibres in equal measure, and evidence that this does occur is forthcoming, for on closer examination of sections of the spinal cord from a case of old-standing thrombotic destruction of the cerebellum, which I reported some years ago, in which there existed a degenerative sclerosis of the direct cerebellar tract in its entire length, I find that they present all the histological characteristics of "retrograde degeneration," and I now feel convinced that the case should be ranged in that category. An instance of "retrograde degeneration" occurring in the ascending antero-lateral tract of Gowers' has also been recorded by Gombault and Philippe. Furthermore, the changes in the nervous system to be found after the amputation

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of extremities, viz., the spinal hemiatrophy, the disappearance of groups of nerve-cells in the anterior cornua which are known to be related to groups of muscles in the amputated part, must be ascribed to a similar process.

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(A) COMMA-SHAPED TRACT OF SCHULTZE AND (B) OVAL CENTRE OF FLECHSIG.

In the *Archiv. für Psychiatrie* for 1883 will be found Schultze's original report of the case of spinal compression in which, in addition to other tracts, this small bundle of fibres in the postero-external columns which now bears that writer's name was found degenerated. Schultze described the bundle as appearing on transverse section in the form of a comma-shaped line in the postero-external column, a line running parallel to the inner edge of the posterior cornu, with its anterior extremity not quite reaching the posterior commissure, and its posterior extremity stopping short some distance off the periphery of the cord. Schultze was unable to follow degeneration in it more than two or three centimetres below the site of the lesion, and supposed that the bundle was composed of descending offshoots of posterior root fibres, a view which we now believe to be incorrect.

It must be mentioned that "kudos" for the discovery of this particular descending degeneration in the posterior columns should not be solely allotted to Schultze, for Bastian, as early as 1867, in a case of injury to the spinal cord in the cervical enlargement mentions such a change, and likewise Kahler and Pick, Strümpell, and Westphal, prior to Schultze, published cases exhibiting analogous degenerations.

More recently detailed descriptions of important cases observed by Gombault and Philippe working conjointly, Barbacci, Tooth, Bruce and Muir, and Hoche, serve to fill up many blanks in the anatomy and connections of this curious tract.

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Tooth's case was one of fracture of the spine with consequent almost complete transverse myelitis between the eighth cervical and the first dorsal segments. Degeneration in comma form could be followed down the cord to between the sixth and seventh dorsal segments, lower, therefore, than in Schultze's case, though the method of Marchi does not seem to have been employed.

Gombault and Philippe, in a lengthy paper on systematised lesions in the white columns of the spinal cord, give a detailed description of this bundle of fibres; they review and criticise the work of previous writers, and fully report valuable cases which throw a flood of light on the matter, and force us to considerably modify our views respecting the regional distribution and connections of the tract. A case of complete sclerosis of the fifth and partial sclerosis of the fourth posterior cervical roots, the change being attributable to Saturnism, is adduced for the purpose of disproving the original view promulgated by Schultze, that the comma-shaped tract is formed by descending offshoots of the posterior root fibres. In the case in question, though the root lesion occasioned the familiar ascending changes above the point of entry of the roots, absolutely no degeneration in the posterior columns below the lesion, such as one would expect if the above-mentioned connection actually existed, was discernible. This view of Gombault and Philippe's does not stand unsupported in the literature, for Tooth previously discredited the connection, observing in the spinal cords of animals, in which the posterior nerve roots had been experimentally divided, that no descending degeneration in the comma of Schultze occurred as a result of the operation.

Two cases of spinal compression, one at the eighth cervical, the other between the fourth and sixth dorsal roots, reported by Hoche, furnish a clue to the termination of this tract. The cords were examined by the method of Marchi, and the fibres of the tract were seen to end above the first appearance of the "oval centre of Flechsig," that is, above the twelfth dorsal segment, by passing into the grey substance of the posterior cornu; and as no other observer has traced the tract lower than this level we must for the present accept Hoche's observations as correct.

We might sum up our remarks on this bundle of fibres by saying that it is a small comma-shaped tract, situated in the postero-external column, which degenerates in a descending direction in consequence of a transverse spinal lesion. It arises in the cervical part of the cord, but in what manner and from what cells is unknown, and it terminates in the grey substance of

the posterior cornu in the lower dorsal region. It is independent of the posterior nerve roots, and is probably a system of endogenous internuncial or commissural fibres.

Hitherto one has only mentioned cases of lesions in the upper dorsal or cervical levels of the spinal cord which have given rise to descending degeneration in the posterior columns. Now there are several most interesting cases on record of lesions at lower levels, which have likewise given rise to a descending posterior column degeneration if anything more pronounced, and the question has arisen whether these descending fibres in the posterior columns in the lower levels of the cord are analogous to the fibres which compose the comma-shaped tract of Schultze in the upper levels. The point has been widely discussed, but as yet no satisfactory conclusion has been arrived at. Here is an epitome of the more important cases. In the first instance Gombault and Philippe, in a case of traumatic transverse myelitis situated at the level of the lumbar swelling, found below Schiefferdecker's traumatic zone, *i.e.*, the zone of complete degeneration, which extends through the breadth of the cord for some few millimetres upwards and downwards from the site of the lesion, descending degeneration in the posterior columns, confined to a wedge-shaped triangular area, situated on each side of the posterior median septum, the apex of the triangle pointing to and reaching the commissure, the base being at the periphery. The extent of the degenerated area proportionately diminished in its downward course, and in sections made at a point immediately above the conus medullaris, the apex of the triangle was situated at a point equidistant between the commissure and the posterior margin of the cord.

Bruce and Muir, in a case of fracture dislocation completely destroying the cord in the upper lumbar region, found by the method of Marchi a similar descending degeneration. At the third lumbar root the diseased fibres became collected along the margin of the hinder portion of the postero-median septum and the periphery of the inner half of the posterior columns. At the level of the fourth lumbar root, the tract extended forward as a narrow band along the side of the septum as far as the posterior commissure. At the first and second sacral roots it had the form of a narrow wedge with its base at the periphery and its apex at the posterior commissure. Below the third sacral root the tract diminished in size as far as the coccygeal segment, but retained its triangular form and its relation to the postero-median septum, and the degeneration terminated by passing forwards into the grey matter at the base of the posterior horn of the same side.

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In Hoche's two cases of spinal compression in the cervical and dorsal regions respectively, mentioned above as showing degeneration of the comma-shaped tract of Schultze, there also occurred a degeneration in the lumbo-sacral region, which, judging from his drawings, is identical with that found by Bruce and Muir, and similar to that described by Gombault and Philippe. The special point of interest in Hoche's cases is that they show what the upper course of this tract of fibres is. One segment below the lesion he describes them as appearing in the most lateral and external part of the postero-lateral column; they then gradually retreat along the periphery until at the twelfth dorsal level they reach the posterior median septum, and then assume the position described above in Bruce and Muir's case.

In 1891 Barbacci, also using the method of Marchi, reported a case of an intra-medullary tubercular focus situated at the level of the sixth and seventh dorsal nerve roots in which descending degeneration was found very much resembling that described by Hoche, and Kahler and Pick and Daxenberger have narrated cases which might be placed in the same category.

These observations, therefore, conclusively prove that in addition to the comma-shaped tract of Schulze there is another tract which degenerates downwards in the posterior columns, one of greater length (at least 23 spinal segments—Hoche), arising in the cervical region (Hoche), and terminating in the grey matter of the conus medullaris (Bruce and Muir, Hoche and Barbacci); but what its precise origin and connections above are, and also whether it is related to the comma-shaped tract of Schultze, is undetermined. Hoche and Gombault and Philippe regard it as a commissural tract of endogenous origin, and although, as Hoche emphasises, the comma-shaped tract of Schultze absolutely terminates in the lower dorsal region, and takes no part whatever in the continuation of the tract, yet Gombault and Philippe's suggestion that the two tracts are analogous is a reasonable one, because the following resemblances exist:—Both are situated in the posterior column, both degenerate in a descending direction in consequence of a transverse lesion, and both are independent of the posterior roots. Gombault and Philippe proved the latter point in connection with the lower tract by quoting a case of tabes, in which the fourth, fifth, and sixth posterior sacral roots were completely atrophied, and this group of fibres remained unaffected by degenerative processes, while the remainder of the posterior columns was completely sclerosed. There seems, lastly, to be no doubt that the "oval centre" of Flechsig in reality represents the lumbar segment of this tract, and that the "dorso-

median sacral bundle" of Obersteiner represents the sacral segment of the tract, and as both these names are as unnecessary as they are cumbersome and confusing, we are quite in accord with Bruce and Muir's suggestion, that because it bears a relation to the posterior median septum, analogous to that borne by the "sulco-marginal tract" of Marie to the anterior median fissure or sulcus, it should be called "the descending septo-marginal tract."[1]

LITERATURE ON COMMA-SHAPED TRACT OF SCHULTZE AND OVAL CENTRE OF FLECHSIG.

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Daxenberger. "Uber einen Fall von chronischer Compression des Halsmarkes." *Deutsche Zeitschr. für Nervenheilkunde*, 1893.

Kahler und Pick. *Archiv. für Psychiatrie*, 1880.

Silvester Blum. "Ueber absteigende secundäre Degeneration in den Hinterstränge des Rückenmarkes." *Inaug. Diss.*, Strasburg, 1895.

[1] As the "oval centre," or "medianen Abschnitt der medialen hinteren Wurzelzone," Flechsig described two small longitudinal bundles, running in the posterior columns of the lumbar enlargement, situated one on each side of the posterior median septum, neither reaching the posterior commissure nor touching the periphery of the cord. The shape of each individual bundle is, on transverse section, plano-convex, with the convexity directed outwards, and the two plane surfaces come into opposition at the median fissure, so that the two bundles when viewed together form a bi-convex or oval field, hence the name "oval centre." The integrity of this field in tabes is well recognised, and furthermore, if in a tabetic cord serial sections be made from the lumbar region to the conus medullaris, one can be assured of a reciprocal connection or continuity between this centre and the so-called "dorso-median sacral bundle" of Obersteiner.

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Intermediary Bundle of Antero-lateral Column (Loewenthal).

This is a scattered tract, the major part of which occupies the mesial portion of the antero-lateral columns in front of the lateral pyramidal tract; but many aberrant fibres are found towards the periphery in the antero-lateral ascending tract of Gowers, and in the direct cerebellar tract and also in the adjoining crossed pyramidal tract. Loewenthal, in papers published in 1885 and 1886, founded on observations of the degenerations resulting from Schiff's experimental operations on the cerebral hemispheres of dogs, assigned to this tract the position named above, and named it the "Système intermédiaire ou faisceau intermédiaire du cordon latéral." He regarded it as a special system of long fibres, and noticing that the fibres composing the tract did not degenerate after removal of the motor area, he argued that they could have no connection with the pyramidal tracts and the motor cortex, such as was claimed by Schiefferdecker and Singer, who noticed degeneration in the tract after sections of the dorsal cord in dogs previous to Loewenthal's description of it. The fibres of the tract are of larger calibre than those of the lateral pyramidal tract, they are clearly seen to degenerate after lesions of the medulla oblongata or spinal cord, they are most abundant in the cervical region, diminishing in number as they descend, and their course is long, as they have been traced into the upper lumbar segments.

Loewenthal was uncertain concerning the point of origin of these fibres, and though some recent papers clear the matter to some extent, there is at the present considerable doubt on this issue. Perhaps the most valuable paper dealing with the subject is from the pen of Boyce. This observer, in an extended series of observations on cats, found that after complete removal of one hemisphere, a bundle of degenerated fibres could be seen at the level of the anterior corpora quadrigemina, crossing in Forel's decussation in the raphe and proceeding outwards to occupy a position behind the inner border of the crusta and close to the mesial and dorsal aspect of the lemniscus on the side contralateral to the lesion. This bundle so formed could be traced downwards in the pons and medulla oblongata, always maintaining a lateral position, until ultimately it gained the lateral column of the spinal cord, along which it could be followed as far down as the lumbar region. The position the tract occupies in the lateral column corresponds closely with the area assigned by Loewenthal to the "système intermédiaire," and Boyce names his

bundle of fibres the "lateral columnar tract." Boyce found that precisely similar degeneration of the tract followed division of the crus cerebri or hemisection of the spinal cord a little below the pyramidal decussation; but there was no degeneration in it subsequent to removal of the motor cortex. On summing up he states that the fibres "are probably the representatives in the medulla, pons, and mesencephalon of the large descending extra-pyramidal fibres found in the lateral columns of the cord, standing in the same relationship to those as do the fibres in the posterior longitudinal bundle and raphe to the anterior and antero-lateral columns. It is possible that in the cat and the dog this higher 'internuncial' or segmental system is more marked than in the monkey or in man, just as in the latter examples the pyramidal system is far more extensive than in the former."

It will be seen that Boyce, though he gives us an admirable description of the tract as it occurs in the cat, offers us nothing more than a clue to its exact origin; and Langley, in telling us that Bouchard in 1866 observed in man that the field of descending degeneration in the lateral column, which resulted from injury to the medulla oblongata, was more extensive than that after injury higher up, and that he located fibres which come from the medulla oblongata in the lateral border of the pyramidal tract, and named them the "fibres commissurales antérieures longues," helps us little, nor does he bring us any nearer the point when in agreement with Sherrington he informs us that the cortical pyramidal tract is of smaller dimensions than that figured by Flechsig, and proposes the name "descending medullary tract" for these extra-pyramidal fibres.

Degenerations consequent on Lesions of the Cerebellum.—Some experimental work completed by Marchi several years back, at first sight adds confusion to our knowledge of the composition and origin of the intermediary system of fibres, but as we now have ample proof that the writer's observations were erroneous, we may fortunately disregard his conclusions. Marchi found that if a hemi-extirpation of the cerebellum of the ape be effected, a secondary descending degeneration ensued in the spinal cord, which corresponded topographically almost precisely to the regional distribution of the "descending sulco-marginal bundle" and the "intermediary system of the lateral column" combined, and the degeneration extended down as far as the lumbar region. He naturally concluded that this system was a descending one, the trophic centre of which was in the cerebellum. Biedl, working in Stricker's laboratory in Vienna, in a paper founded on a

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series of observations on cats, of which he had divided the corpus restiforme, writes in confirmation of Marchi's experiments, and so likewise does Thomas. Three English observers, however, Ferrier and Turner, who worked conjointly, and Risien Russell, are entirely opposed to these views originated by Marchi. Having performed many experiments similar to these initiated by Marchi, they are unanimous in denying the occurrence of descending degeneration in the spinal cord as a sequel to an experimental cerebellar lesion in the lower animals, and firmly express the opinion that the degeneration which Marchi obtained was in reality due to an unintentional wounding of Deiter's nucleus and other structures in the lateral region of the medulla, an accident which it would appear it is difficult, nay, almost impossible to avoid during the operation of cerebellar hemi-extirpation, owing to the proximity of the nucleus to the inferior cerebellar peduncle or restiform body, and an injury which is sufficient in itself to produce the exact spinal degeneration figured by Marchi.

This controversy, therefore, has not only been of value in disproving the existence of a direct descending cerebello-spinal tract, but has also led to a closer definition of the origin of Loewenthal's tract, for Risien Russell, who has studied the matter more laboriously than any other observer, in his last paper arrives at the following conclusions :—That the real source of origin of the descending antero-lateral tract, which degenerates in the spinal cord after a lesion of the lateral region of the medulla oblongata, is, as Ferrier and Turner agreed, Deiter's nucleus; that the degenerated fibres, which reach the anterior columns of the upper part of the spinal cord after a lesion of Deiter's nucleus through the posterior longitudinal bundles, are quite distinct from the above tract, and probably belong to some system of internuncial fibres similar to those traced by Boyce to the anterior columns by way of the posterior longitudinal bundles, after hemisection of the mesencephalon ; and lastly, that the direct descending tract of degenerated fibres met with in the spinal cord, in close relationship to the fibres of the crossed pyramidal tract, after a lesion of the lateral region of the medulla, is probably identical with the tract described by Boyce after hemisection of the mesencephalon.

An example of the perplexity occasioned by acceptance without confirmation of Marchi's observations is afforded by a recent paper of Bechterew's. This writer describes a system of fibres equally scattered throughout the lateral pyramidal tracts, which is to be observed in the spinal cord of a fœtus at a time when the ordinary pyramidal fibres are still unmyelinated, and which are untouched in an ordinary secondary descending de-

generation, and on the strength of Marchi and Biedl's observations, he states that they degenerate after idio-lateral cerebellar hemi-extirpation, but it is much more probable that they are aberrant fibres of Loewenthal's intermediary system.

LITERATURE ON INTERMEDIARY BUNDLE OF ANTERO-LATERAL COLUMN (LOEWENTHAL).

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BOYCE. *Loc. cit.*

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DESCENDING SULCO-MARGINAL BUNDLE (MARIE).

"Système descendant de la zone sulco-marginale" is the name affixed by Marie to a scattered group of fibres, situated in the anterior columns along the margin of the anterior fissure, which undergo degeneration in a downward direction in consequence of a transverse spinal lesion. The length of these fibres

varies; some have been traced from the cervical to the lumbar segments. Though they possess a similar distribution, they are not, as Schiefferdecker thought, aberrant fibres of the direct pyramidal tract, for they do not degenerate after cerebral lesions, and are further to be found in the lower segments of the cord, where the direct pyramids are not represented (Flechsig, Singer). In the dog Singer and Münzer place the fibres of this tract in a small group at the anterior angle of the cord, opposite the mouth of the anterior spinal fissure. In the cat it occupies a similar position, and Schäfer has shown that in that animal this bundle is one of the earliest to undergo secondary degeneration, its fibres exhibiting the characteristic reaction with osmic acid on the fourth day after a spinal hemisection.

Marie thinks it probable that these fibres appertain to the system of longitudinal or internuncial fibres, the existence of which Ramon y Cajal and Golgi have so beautifully demonstrated, and that, originating from an upper cell of the anterior cornu, they cross by the anterior commissure, and course downwards to connect that cell with one situated in the opposite anterior cornu of a lower segment of the cord. We fear that there is insufficient anatomical evidence for the existence of the anterior commissural decussation which Marie alludes to, and since destruction of Deiter's nucleus gives rise to descending degeneration of fibres situated in the sulco-marginal zone, indistinguishable from Marie's fibres (*vide* last paragraph), we would rather assume that the trophic centre for this system of neurons is situated in that nucleus or thereabouts. In man the bundle demands further investigation.

Postero-external Column or Column of Burdach, and Postero-internal Column or Column of Goll.

Since the fibres which compose these columns are derived from a like source, and since Goll's column is really made up of overflow fibres from Burdach's column, it is deemed advisable, as well as convenient, to consider the two together.

Within the past few years these columns have been exhaustively studied by a host of competent observers, some describing the changes in them consequent on division of the posterior nerve roots or spinal hemisections in lower animals (Tooth, Horsley, Mott, Sherrington, Singer and Münzer, Barbacci), others reporting the degenerations attendant upon traumatic lesions or tumours.

&c., of the cord or posterior nerve roots (Schiefferdecker, Gombault and Philippe, Déjérine), and others studying the alterations occurring in them as a result of various diseases (Mayer, Redlich, Obersteiner). In short, our knowledge of these two great centripetal tracts has been increased by leaps and bounds, and at the present time is practically complete, with the exception that the ultimate distribution of the tracts after the fibres quit the nuclei of Goll and Burdach still requires further elucidation.

Development.—Flechsig's development researches show that the fibres of the column of Goll become myelinated at the end of the sixth month of fœtal life, and thus are among the earliest fibres to assume the medullary envelope; and Schäfer in his experiments on cats found that the fibres of the column of Goll degenerated on the fourth day after a hemisection of the cord, and was one of the first tracts to yield evidence of the secondary changes. There is, therefore, a parallel between the time of accession of the secondary degeneration and the period of development of the medullated sheath in this column.

Origin and Destination.—From the degenerations observed after section of the posterior nerve roots, and from diseased conditions affecting these roots, numerous writers have demonstrated that the fibres composing these columns represent the spinal continuations of these posterior roots—in short, they are exclusively exogenous fibres. Although a side issue, it should here be indicated that in certain of the lower animals, such as the frog, chick, &c., a certain number of fibres having an intra-spinal origin issue in the posterior root, and hence do not degenerate in consequence of a root section (v. Lenhossek, Ramon y Cajal, Horton Smith), and the question has arisen whether analogous fibres do not exist in mammals and the human being. But in the case of the cat and the monkey Sherrington, v. Lenhossek and Ramon y Cajal's observations definitely negative this idea, and therefore the original law of Bell, which states that the posterior roots exclusively consist of fibres of extra-spinal origin destined for the conduction of sensory impressions, certainly holds in the case of some mammals, and probably also in the human being.

The fibres of the posterior roots enter the cord in that region which is situated behind the substantia gelatinosa Rolandi, and the most laterally situated fibres of this portion form Lissauer's so-called rootzone. It has been noticed by Obersteiner that just at the point of entry into the cord each fibre dispenses with its medullated sheath, but only for a very brief distance. From personal observations I am convinced of the correctness of this statement, and may mention that this gap in the medulla of the

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fibres may be most clearly seen if an acutely degenerated posterior root be stained by the method of Marchi.

Having gained their entry into the spinal cord, the fibres divide into ascending and descending branches, each of which issue collaterals in their course. The descending branches pass downwards for a short distance, and then turn at right angles into the grey matter at the top of the posterior cornu, where they end in arborisations round nerve cells. The ascending branches have varying destinations, and either by means of their terminal branches or their collaterals are said to come into relation with a number of different groups of nerve cells; certainly many fibres go to (*a*) cells in the middle segment of the posterior cornu; others seem to be destined for (*b*) the motor anterior cornual cells, and form the reflex collaterals of Kölliker or postero-anterior collaterals of Ramon y Cajal; others proceed to (*c*) the cells of Clarke's column, and are seen atrophied in tabes dorsalis; a small number of collaterals cross in the grey comissure and go to (*d*) cells in the posterior cornu of the opposite side; and, lastly, the long fibres of the columns reach (*e*) the cells of the nuclei of Goll and Burdach. The researches of Ramon y Cajal, v. Lenhossek, Golgi and Kölliker seem to prove that these fibres, when they approximate these various groups of nerve cells, break up into arborisations which surround the bodies of the cells without visibly coming into contact with them or penetrating their interior; so that if a recently degenerated nerve fibre be followed up to one of the nerve cells, delicate methods may display a disappearance of the investing arborisation, while the cell body and its processes remain intact. But that this cell body or "Endkerne," as His calls it, so shorn of its investing diminutive nerve plexus, undergoes degeneration and disintegration at a later stage is almost certain.

In addition to the above-mentioned destinations of the posterior root fibres, others are supposed by Loewenthal, Oddi and Rossi and Berdez, on the strength of experimental researches, to pass by way of the anterior commissure either into the opposite posterior column or to enter one of the long ascending tracts of the anterior or lateral columns, but the existence of such a decussation is denied by more recent observers. Fajersztajn asserts that the posterior roots have no connection whatever with the anterior commissure, and bases his statement on convincing deductions drawn from experiments on animals; these were the isolation of lengths of the spinal cord by two complete transverse sections made at distances of 6-14 centimetres from one another, and in these cases he found the anterior commissure perfectly

free from degeneration in the neighbourhood of the lower hemisection, where all ascending fibres must necessarily be degenerated. Souques and Marinesco and Mott share this opinion, and the latter mentions the supposition that if degeneration occurs in the crossed afferent tracts of Gowers and Edinger (*vide* antero-lateral ascending tract) it might be attributed, not to the section of the posterior roots, but to unavoidable vascular injuries incident to such an operation affecting nerve cells in the grey matter of the posterior horn, from which it is believed the fibres of the crossed afferent tract of Edinger originates. These latter fibres, it must be noted, are supposed to cross in the anterior commissure.

Lastly, certain physiological experiments point to the probability of a decussation of fibres, either by way of the grey commissure, or directly through the posterior median septum to the opposite side, but no anatomical basis exists for this supposition.

Topography.—Having now indicated the latest views concerning the destinations of the posterior root-fibres, it will next be necessary to refer in some detail to the topographical distribution and arrangement of the individual groups of posterior root-fibres, commencing with those derived from the cauda equina.

Excellent cases, illustrating the course of the degeneration which follows destruction of the cauda equina in the human being, have been recorded by Souques and Marinesco, Darkschewitch, and Déjérine and Sottas; and the following descriptive *résumé* of Souques and Marinesco's case applies more or less to all, and may be accepted as typical. In this instance the cauda equina and conus medullaris, and the three lower sacral segments, were destroyed by the pressure of a group of hydatid cysts. The middle sacral segments exhibited the changes of traumatic degeneration (Schiefferdecker); in the upper sacral segments the entire posterior columns, with the exception of the centrum ovale (Flechsig), the cornu-commissural zone (Marie), and some few fibres coursing along the inner margin of the posterior horns, were degenerated; likewise the delicate myelinic plexus, situate in the grey matter of the posterior and anterior cornua, was at this level markedly atrophied. In the mid-lumbar region similar changes were visible. In the lower dorsal region the degenerated area was separated from the posterior cornua by a definite intervening zone of healthy fibres, and the myelinic plexuses in the anterior cornua were healthy. In the mid-dorsal region the plexus of nerve fibres in the posterior cornua regained a healthy appearance, and the patch of degeneration assumed the form of a funnel or wedge, the point of which reached the posterior commissure, while the base lay

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along the periphery. In the upper dorsal region the degeneration became limited to the postero-internal columns, and retreated from the commissure. In the cervical region it affected only the posterior half of the postero-internal columns, and in the medulla degenerated fibres were traceable to the inner side of the nucleus of the funiculus gracilis. Lastly, it should be added that the total volume of the degenerated fibres gradually diminished as the diseased field ascended.

Tooth obtained degeneration resembling this in its distribution, after division of the posterior roots of the cauda equina in the monkey; and Mott, after section of the lumbo-sacral roots in the monkey and cat, got confirmatory results.

The experiments performed by Tooth of division of all the posterior nerve roots between the third dorsal and the third lumbar segments, inclusive, produced like results. The patch of degenerated fibres was situated immediately external in position to that above described, and likewise gradually spread inwards, until in the upper dorsal region it was almost confined to the postero-internal column; then, as it extended along the cervical region, it gradually retired from the posterior commissure, while maintaining its peripheral base. The degenerated fibres terminated mainly in the outer part of the nucleus of the funiculus gracilis.

In the case of the first and second dorsal nerve roots and the two lower cervical roots, some of their fibres pass over to the postero-internal columns, but a portion do not leave the column of Burdach, and in regard to the remaining upper cervical nerve roots, their fibres canton for their entire course in the postero-external column, gradually taking up a more internal position as they ascend, and ultimately gaining the nucleus of the funiculus cuneatus.

A case narrated by Gombault and Philippe, in which there was complete destruction of the fifth right posterior cervical root, and partial destruction of the fourth posterior cervical root on the same side, beautifully illustrates the degeneration as it occurs in the cervical region in the human being. Immediately below the fourth cervical roots, the patch of degeneration is triangular in shape, approximated to the posterior part of the cornu, and separated from the periphery by a thin band of healthy fibres. At the level of the first cervical roots it is seen as a strip, extending from the periphery to the posterior commissure along the inner margin of the postero-internal column. It occurs as a similar strip in the funiculus cuneatus at the first cervical level, and eventually becomes localised in the cuneate nucleus.

From these accounts it may readily be gathered that the long fibres derived from the posterior roots possess one remarkable peculiarity in regard to the course which they pursue, viz., that as they ascend they are gradually being pushed into a more internal position, and having the position which they have just vacated occupied by fibres which enter from the root immediately above. In this manner it comes about that those fibres which occupy the external division of the posterior field in the lower segments of the cord are found in the internal division in the upper segments. This assumption of an eccentric position by the long fibres of the posterior roots is one which is common to a number of other systems, and has led Flatau to formulate the law "that the short fibres of the cord run in close position to the grey matter, while the long fibres select a position next the periphery."

Numerous other reports of the degenerations which have ensued after various forms of transverse lesions in different parts of the spinal cord, and many other experimental researches, might be cited in illustration of the topographical distribution of the posterior root fibres. But this is hardly necessary, and it may be taken for granted that the sum total of these observations is (1) that the fibres which constitute the postero-internal column or column of Goll and eventually enter the nucleus funiculus gracilis, are derived from the posterior roots of the sacral and lumbar segments and of the dorsal segments, with the exception of the first and second, and that all the fibres which enter into the composition of this tract have in one part of their course cantonned in the column of Burdach; (2) that those fibres which constitute the postero-external column or column of Burdach, and terminate in the nucleus funiculus cuneatus, are derived from the posterior roots of the cervical segments, and partly from the two upper dorsal posterior nerve roots.

Distribution of the fibres which emanate from the nuclei of the funiculus gracilis and funiculus cuneatus.

As already stated, considerable doubt exists concerning the ultimate distribution of the fibres issuing from Goll's and Burdach's nuclei, or the fibres of the second sensory neuron as they might be called; the reason, of course, being that lesions which will cause an interruption of these secondary neurons alone, without involving other tracts, are rarely met with. However, the accepted view is, that these fibres are destined for the cortex of the cerebrum and cerebellum respectively, that they are

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conveyed to the former in the fillet or lemniscus, to the latter by the corpus restiforme or inferior cerebellar peduncle.

In the first place, in regard to the fillet, articles sufficient to form a considerable volume have been published bearing on its course and connections, but it will be impossible in this review to do more than outline some of the more important observations on the tract.

To begin with, some experimental anatomical papers dealing with the course of these fibres are of interest. Mott, after unilateral separation of the nuclei of the posterior columns from the arciform fibres issuing therefrom in the monkey, obtained degeneration of these fibres and of the opposite inter-olivary layer. In the upper part of the pons varolii the degenerated fibres occupied the median and lateral fillet; some fibres of the lateral fillet apparently went to the corpora quadrigemina, but the majority of the remaining fibres could be traced onwards to the optic thalamus, where they terminated. Evidence of a direct continuity of these degenerated fibres by a "cortical fillet" with the cortex cerebri was not obtainable.

Likewise Singer and Münzer, by destroying the cuneate nuclei, obtained atrophy of the internal arciform fibres of the same side and of the fillet of the opposite side, which was traceable to the optic thalamus.

von Vejas also years ago discovered that destruction of the posterior column nuclei of new-born animals induced atrophy of the same part of the fillet as far as the corpus trapezoides.

Schlesinger, in a most thorough paper on degenerations of the lemniscus well worthy of consultation by anyone specially interested in this tract, traces some fibres of the posterior column nuclei to the anterior corpora quadrigemina and the majority to the optic thalamus. All Schlesinger's observations were made on the human being.

From the foregoing it might be assumed that these fillet fibres issuing from the nuclei of Goll and Burdach's column degenerate only in an ascending direction, but this is not so, for there is an opposite array of evidence to show that they may degenerate or atrophy in a descending direction. Jakob, Mahaim and Bruce have reported such a descending degeneration in cases of central cerebral lesions, and the writer, in a case of tubercular softening of the optic thalamus, examined by the method of Marchi, saw descending degeneration in this tract.

Therefore, though confusing, it seems, nevertheless, to be correct to assume that the fibres of the fillet may degenerate either upwards in consequence of an interruptive lesion near the medul-

lary nuclei, or downwards in consequence of a lesion of the great nuclei at the base of the brain.

Now, since an interruptive lesion of the fillet gives rise to a degeneration which cannot be traced higher than the optic thalamus, the question is, how are sensory impulses conveyed further to the cortex cerebri? And the supposition is that between the thalami and the cortex a third neuron is trajected, which has been named by some the "cortical fillet," and which may under certain conditions undergo degeneration. For instance, Monakow, by destroying the parietal lobe in young animals, produced atrophy of the fillet of the same side and of the internal arciform fibres and nucleus gracilis. Flechsig and Hosel saw the same changes in a case of porencephaly involving the central convolutions; similar cases to this have been recorded, and in two cases of cerebral lesions above the level of the basal nuclei, I have noted corresponding atrophy of the fillet.

We may then sum up by saying that the fibres issuing from the nuclei of Goll and Burdach's columns, which are destined for the cortex cerebri, ascend in the lemniscus or fillet. These fibres decussate in the medulla, continue along the pons and crus cerebri in the mesial and lateral lemniscus, and after giving off collaterals to various nuclear structures, amongst others the anterior corpora quadrigemina and the corpora Luysii, they eventually reach the optic thalamus. At this level it is supposed a third system of sensory neurons begins, the axons of which are first grouped in that part of the posterior limb of the internal capsule which Charcot has named the "carrefour sensitif," and thence stream into the corona radiata to terminate in the cortex of the parieto-occipital and temporal convolutions. But more information on this system of neurons is needed.

Secondly, one must briefly refer to the connection between fibres issuing from the posterior column nuclei and the opposite lobe of the cerebellum. According to Monakow the fibres issuing from the cells of the outer or lateral part of the nucleus of Burdach's column are specially destined for the cerebellum; these fibres are joined by others from the nucleus gracilis, and are directed to the restiform body by the internal arciform fibres, and hence reach the cerebellum, but what part thereof is not exactly known.

According to Bechterew the nucleus gracilis has another crossed connection with the cerebellum; this writer states that some few of the fillet fibres at the decussation of that structure, instead of continuing upwards, pass forwards through the opposite anterior pyramid, and reaching its ventral surface, curve round

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the periphery of the medulla oblongata as external arcuate fibres to the corpus restiforme, and thence reach the cerebellum.

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LISSAUER'S ROOTZONE, OR COLUMN.

As Lissauer's rootzone, Waldeyer's "Markbrücke," or Flechsig's "laterale hintere Wurzelzone," is recognised that part of the cord which on transverse section lies immediately dorsal and lateral to the apex of the posterior cornu. All that we know about the area is that it contains fibres of small calibre, supposed to be derived from the posterior roots, and that they proceed in through the substantia gelatinosa to cells of the posterior cornua. Lesions which cause ascending degeneration in the posterior columns also

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give rise to degeneration of Lissauer's column, and it is invariably sclerosed in tabes dorsalis. Bechterew has indicated that the fibres of this column are peculiar in becoming myelinated at a later period than any other of the posterior root fibres.

Direct Cerebellar Tract.

The name given to this important afferent tract by Flechsig, its original describer, was "the direct lateral cerebellar tract," but it is now occasionally called the "dorso-lateral, or postero-lateral, ascending cerebellar tract," in contradistinction to the "ventro-lateral or antero-lateral ascending cerebellar tract," *i.e.*, the antero-lateral ascending tract of Gowers.

Development.—The fibres of the tract acquire their medullated sheath not later than the beginning of the sixth month of fœtal life (Bechterew), which is considerably prior to the period of medullation of the fibres of the antero-lateral ascending tract.

Origin.—In regard to the origin and constitution of this tract, we are, as Marie says, even at the present time, in a condition of absolute uncertainty. The two main views in reference to its origin are (1) that the fibres are derived from the cells of the vesicular column of Clarke, a view championed by Mott; (2) that they are derived from the posterior roots, a mode of origin credited by Tooth.

Mott describes how "the cells of the column of Clarke give off large fibres which run upwards, slightly forwards, and then outwards through the lateral column, to reach the direct cerebellar tract of the same side." And while admitting that on account of the obliquity of the course followed by these fibres between their starting-point in the vesicular column and their cantonment in the direct cerebellar tract, it is impossible to cut a section which will include a view of the fibre in the whole of this part of its course, yet he adduces a quantity of evidence—histological, developmental, and experimental—in favour of his claim. Perhaps the most important experiment in support of the view was that of unilateral section of Clarke's column, effected in a monkey at the level of the twelfth dorsal and first lumbar segments, which resulted in ascending degeneration of the direct cerebellar tract on the side of the lesion. Sherrington acquiesces in Mott's views, and they are further supported by Flechsig's developmental researches.

Touching the second theory of origin, Tooth agrees with Mott in thinking that the fibres of the tract do not come by way of the

posterior roots for the lower limbs, but he found that if in monkeys he divided the 6th, 7th, and 8th cervical and the 1st dorsal posterior nerve roots, a complete and absolute degeneration—not of the whole direct cerebellar tract, but of that part of it which lies next the posterior horn—occurs. Like results followed section of the 2nd, 3rd, and 4th dorsal posterior roots. He therefore concludes that certainly in the cervical region, and probably also in the upper dorsal region, fibres of the posterior roots enter largely into the composition of the tract. It is to be further gathered from Tooth's work that he regards the direct cerebellar tract as being entirely made up of fibres of large calibre (Bechterew supports this statement), and that the fibres of small calibre seen in the position of the tract in the lower dorsal region, are descending ones, probably pyramidal fibres, as the pyramidal tract comes to the surface at this level, and that the fibres which Mott and others have seen obliquely crossing the lateral column at this level are fibres of this nature. Tooth's conclusions are rendered insecure by the fact that Singer, Kahler, and others, in their experiments of section of the posterior spinal roots of the dog, did not obtain degeneration of the direct cerebellar tract.

We should also like to ask why it is the exception and not the rule to find sclerosis of the direct cerebellar tracts in cases of tabes dorsalis, in which disease the posterior roots are completely withered up by sclerotic processes.

Unfortunately the recently recorded observations of two Italian experimental neurologists, Pellizzi and Paladino, are not reconcilable to either of the above-mentioned views, and indeed are altogether extraordinary as well as out of accord with the observations of other better known observers, that is, provided the anatomical distribution of the fibres of the spinal cord be similar in the dog to what it is in the monkey, as we may presume it is.

Pellizzi, after unilateral division of the posterior roots of the last lumbar segment and of the first posterior sacral root, and likewise after a hemisection of the spinal cord at this level, obtained, along with other changes, symmetrical ascending degeneration of both direct cerebellar tracts. A like result followed unilaterial section of the four lower posterior cervical roots. He surmises that the direct cerebellar tract receives its fibres partly from the column of Clarke of the same side, partly from the posterior roots of the same side direct, and partly from the posterior roots of the opposite side, the fibres of the latter decussating in the anterior commissure.

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Paladino, after unilateral section of the sixth posterior roots of the lumbo-sacral plexus, also obtained bilateral ascending degeneration of the direct cerebellar tracts, and a similar result after unilateral division of the four lower dorsal posterior roots.

Both workers stained their sections by the method of Marchi. Results such as these render our confusion concerning the origin and composition of this tract more profound, but judging from the work of previous experimenters and a quantity of other evidence, we can hardly seriously accept Pellizzi and Paladino's observations as correct, that is to say, as far as the point under consideration, viz., the source of these fibres, is concerned. A difference between the constitution of the cord of the dog and that of the cat or monkey is too unlikely to account for these results, and to my mind they are altogether insufficient to controvert the records of careful experimenters, like Mott and others, who, severing the same roots and employing the same staining method, failed to obtain any degeneration whatever in either direct cerebellar tract, furthermore they are out of harmony with the changes observed in the human cord following lesions of the posterior roots of the lumbo-sacral plexus.

While offering all due respect to Pellizzi and Paladino for their work (Pellizzi's paper is an especially complete one), we therefore feel forced to disregard their conclusions, and hazard the opinion that the anomalous degenerations which their experiments brought forth are to be attributed to some operative mishap, possibly some unnoticed vascular injury, which it is difficult to avoid in these procedures, and which is apt to render any experimental work unreliable.

After sifting all the evidence concerning the origin of the direct cerebellar tract, we are inclined to abide by Mott's decision that it is composed of the neuraxons of the bipolar cells of the vesicular column of Clarke.

Topography.—The level at which this tract commences as a bundle varies with different authors. Tooth concludes from his researches that it begins to show itself somewhere between the 9th and 11th dorsal nerve roots. Mott, and likewise Kahler and Pick, fix it at the 9th dorsal pair, Schultze at the 10th, and more recently Barbacci has observed degeneration in it as low as the point of origin of the 11th and 12th dorsal pair of nerve roots. It is probable that the site at which it appears will vary in different individuals in the same way as the downward distribution of the columns of Clarke is inconstant.

Since it is an established fact that Clarke's column commences to appear at the 2nd or 3rd lumbar segment, and since the direct

cerebellar tract is said to be derived from these cells, the question may be asked: Why is it that after a lesion in the lumbar region where Clarke's column is already formed, degeneration in the direct cerebellar tract does not present itself until the level of origin of the 11th or 12th dorsal nerve roots is reached? This apparent incongruity is explained, as Barbacci and Mott have demonstrated, by the great obliquity of traject of the fibres as they course from Clarke's column to their eventual seat in the tract. This obliquity is such that though Clarke's column may be represented as low down as the 2nd lumbar segment, the axis cylinders of the nerve cells of the columns do not become grouped in their position in the direct cerebellar tract until a point two or three segments higher up is reached.

In form the direct cerebellar tract has been compared to the segment of a ring, occupying about one-sixth of the spinal circumference. Its posterior extremity is thickened and comes into contact with the tip of the posterior cornu. Its anterior extremity is attenuated and at the same time not sharply defined, its fibres mingling with fibres of the adjoining antero-lateral ascending tract. Throughout the whole extent of its course in the cord the tract more or less maintains this shape and position, but at about the level of the 3rd cervical roots it becomes separated from the antero-lateral ascending tract by the interposition of a collection of heterogeneous fibres.

Higher Distribution and Destination.—As the tip of the posterior cornu quits the periphery in the lower part of the medulla the direct cerebellar tract takes its place; it is thrust further back, a little higher up, by the so-called ascending root of the trigeminal nerve, which inserts itself in front of it. Higher still the tract proceeds, first externally to the ascending 5th root, and then comes to lie anteriorly to it. At the level of origin of the 8th nerves the fibres pass obliquely into the corpus restiforme. Thence Loewenthal, in experiments on dogs, traced it to the dentate nucleus of the cerebellum, and inferred that it ultimately terminated in the superior vermiform process. Tooth believes that it is only after section of the posterior roots of the upper segments of the cord that a simple uncomplicated degeneration of the direct cerebellar tract can be satisfactorily traced upwards, because in hemisections of the cord there is a confusing admixture of degeneration of the antero-lateral elements. Bechterew, however, in developmental observations on the fœtus from 25-27 centimetres long, confirms the statement that the direct cerebellar tract passes to the superior

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vermis, and Monakow and Mott arrive at the same conclusion experimentally.

Retrograde Sclerosis or Degeneration in the Direct Cerebellar Tract.—In 1894 I reported a case of old standing thrombosis of the posterior inferior cerebellar artery which had left a deep and extensive excavation, involving the parts supplied by that artery. Microscopic examination of the cord in this instance revealed profound sclerosis of the direct cerebellar tract occurring throughout the whole extent of this tract, and this was accompanied by atrophy of the cells of Clarke's column, all on the side of the lesion. Now, the only rational conclusion is that the case was one of retrograde sclerosis in consequence of an obliteration of the terminus to which the fibres of the direct cerebellar tract run. More recent publications show that such a retrograde sclerosis may occur in other tracts and so confirm the view.

Pellizzi, after a lesion at the lower dorsal level, and after unilateral section of the four lower posterior cervical roots, obtained slight descending degeneration in the direct cerebellar tract on the side of the lesion. A result, so far as one can gather, never obtained by other experimenters. He explained the degeneration by supposing that the degenerated fibres are descending branches of the posterior roots.

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Antero-lateral Ascending Tract, Ventro-lateral Ascending Cerebellar Tract, or Tract of Gowers.

This is another important tract which degenerates in an ascending direction in consequence of a transverse spinal lesion, and which has received a lot of attention from those workers who have

studied the direct cerebellar tract, with which it was formerly confused. As will be presently indicated, the cerebellum seems to be its destination, and on that account some writers *e.g.*, Mott, Loewenthal, Schäfer, prefer to name it the "ventro-lateral or antero-lateral ascending cerebellar tract." By Continental writers it is generally called after its original discoverer, "Gowers' Tract."

Origin.—As was the case with the "direct cerebellar tract," so is it with the antero-lateral ascending tract, the exact source of the fibres which compose it is uncertain—perhaps to a greater degree—and the views concerning its origin, whether formulated by experimental or purely anatomical workers, are mainly based on conjecture. That the fibres which constitute the tract enter the spinal cord by the posterior roots would appear to be in a measure negatived by the experiments of division of these roots practised by Mott, Tooth and many others; but Pellizzi, whose extraordinary results have already been referred to in the section on the direct cerebellar tract, obtained symmetrical ascending degeneration in both antero-lateral ascending columns after unilateral section of the last lumbar and the first sacral posterior roots of the dog; he also saw slight degeneration of the same tract, but more marked on the side of the lesion after unilateral section of the four lower posterior cervical nerve roots, and naturally concludes from these experiments that each antero-lateral ascending tract receives some of its fibres from both posterior roots, the anterior commissure serving as the bridge of decussation.

Likewise Loewenthal, Berdez and Langley and Anderson, after similar operations on various animals, obtained degeneration either in the anterior commissure or in both antero-lateral ascending tracts, but always more marked in the latter on the side of the lesion.

Also Mott, in a recent paper, states that in one monkey, of which he had divided the fourth, fifth, sixth, seventh, eighth and ninth sub-thoracic roots on one side, he found some thirty degenerated fibres in the opposite antero-lateral ascending tract, but he firmly expresses the belief that this degeneration was set up by vascular injuries to the grey matter of the posterior horn, accidentally induced by the operation, and in effect totally denies the passage of fibres from the posterior roots directly across the cord to the opposite antero-lateral ascending tract. Mott, in the same paper, further mentions that Loewenthal and Berdez's experiments are on similar grounds not to be relied on, for they certainly did not exclude injury of the tip of the posterior horn and contiguous parts, nor did they take into account the effect of such

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injuries. For a like reason Langley and Anderson's experiments cannot be accepted, and it is more than probable that Pellizzi's experiments must be condemned on account of the same flaw.

Unfortunately, we can hardly compare the observations of Kahler, Singer and Münzer and Tooth, or any other experimenter who worked before the method of Marchi was introduced, with the observations of the more recent writers quoted above, all of whom worked with this method, for the simple reason that the method of Weigert and its modifications, which a few years back was the fundamental staining *technique*, fails to reveal degenerated fibres which are disseminated or present only in small groups, whereas the method of Marchi will plainly discover such degeneration.

To support the assumption that no fibres enter the antero-lateral ascending tract directly from the posterior roots, we are therefore practically left with Mott's view only, but this view we prefer to accept until an explanation is forthcoming, showing why it is that in certain cases in which the operation of division of the posterior roots is carefully performed, and the sections of the spinal cord stained by the most recent and approved methods and closely examined, no degeneration whatever is discoverable in either ascending antero-lateral tract. It should also be mentioned that Mott's view is strengthened by the fact that in several cases of injury to the cauda equina in the human being, in which the cord was stained by the method of Marchi, no degeneration whatever was found in either ascending antero-lateral tract (Darkschewitch, Souques and Marinesco, Déjérine and Spiller).

What, then, is the source of these fibres? Tooth, whose opinion deserves consideration in this connection, suggests "that the antero-lateral fibres arise solely from the grey matter, that its fine fibres, which are very numerous in the lower cord, are centripetal from the posterior vesicular column, and that the large fibres are in connection with one or more groups in the anterior horns," but this suggestion, he admits, is quite hypothetical. Hadden and Sherrington's case of tabes, in which the antero-lateral ascending tract was sclerosed, as well as the posterior column, which Tooth quotes in favour of the view that the columns of Clarke give origin to fibres of the antero-lateral ascending tract, does not appear to us to be of much importance, because, though atrophy of the columns of Clarke is almost constant in locomotor ataxia, sclerosis of the lateral columns is rare, and when it does occur involves the direct cerebellar tract as often as, if not more frequently than, the antero-lateral ascending tract. In specimens in which we have seen such changes, we

have attributed them to peripheral vascular alterations concurrent with syphilis and tabes (an idea suggested by Déjérine years ago), rather than to a primary atrophy of Clarke's column.

As far as we can gather, Tooth and Testut appear to be the only authors who maintain that the antero-lateral ascending tract takes any fibres from the columns of Clarke, but there is more or less unanimity of opinion that cells in other parts of the grey matter, particularly at the base of the anterior horn, give origin to it (Bechterew, Mott, Flatau, Gombault and Philippe). Some experiments of Mott's are of considerable importance in this connection; he practised median section of the spinal cord in the lumbar region in two monkeys. In the first experiment the lesion involved the lower end of Clarke's column on one side, and the result was symmetrical ascending degeneration of both ascending antero-lateral tracts, and degeneration of the direct cerebellar tract on the side of the lesion. In the second experiment the incision was successfully made in the middle line below the position of Clarke's column, and while precisely similar bilateral degeneration occurred in the antero-lateral ascending tracts, there was practically none in the direct cerebellar tract. Similar results were obtained by Grünbaum after longitudinal division of the cord. Likewise Ehrlich and Brieger, and Singer and Münzer, finding that compression of the abdominal aorta in rabbits causes destruction of the nerve-cells in the grey matter of the lumbar cord, and also ascending degeneration of the antero-lateral ascending tract, infer therefrom that this tract arises from cells in the grey matter of this region.

These experiments, therefore, in a measure, prove that the fibres of this tract do not originate from the nerve cells of Clarke's columns, but from cells elsewhere in the grey matter whose exact situation is unknown, but supposed to be at the base of the anterior horn, possibly the middle cell column of Waldeyer. The occurrence of bilateral degeneration of the tracts in question after a mesial longitudinal spinal section, warrants the assumption that these cells give off fibres which decussate in the anterior commissure, and so reach the lateral tract, and the existence of such a decussation is also supported by Auerbach's experiment of destruction of a considerable length of one-half of the spinal cord in the cat, which resulted in ascending degeneration of the tract on the side opposite to the lesion.

Crossed Afferent Tract of Edinger.—Some other fibres ascending in the position of the antero-lateral ascending tract must not be confused with those which I have just described; these are afferent fibres which Edinger and Gowers, and likewise Mott,

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believe enter from the posterior roots to come into connection with cells of the grey matter, which cells give off axis cylinder processes, also decussating in the anterior commissure, and also ascending in the region of the antero-lateral ascending tract; coursing along the medulla oblongata and pons in or near the fillet, these fibres terminate in the corpora quadrigemina or optic thalami. This tract so formed is called the "crossed afferent tract of Edinger," and differs from the antero-lateral ascending tract probably in its origin, and certainly in its destination. From what Mott says, it is probable that it was degeneration of this tract, and not of the antero-lateral ascending tract, which Loewenthal, Berdez, Langley and Anderson and Pellizzi saw in their posterior root experiments; also that that degeneration occurred not in consequence of the root section, but subsequent to accidental injury of the most dorsal segment of the posterior cornu, and therefore that the seat of origin of these fibres is in that segment of the posterior cornua.

Topography and Termination of Antero-Lateral Ascending Tract.—Having now pointed out the different views concerning the origin of this tract, I will next proceed to deal with its spinal topographical distribution. Firstly, as to the lowest level in the spinal cord at which it has been found. This level is considerably below that at which the direct cerebellar tract first shows itself; as I previously mentioned, degeneration in that tract has not been definitely seen below the 10th dorsal segment, but there are several cases on record of lesions lower than this which caused degeneration of the antero-lateral ascending tract and left the direct cerebellar tract intact; such, for instance, as Schultze's case, in which there was crushing of the lumbar region. Furthermore, Bechterew mentions the existence of the tract even in the lower parts of the lumbar enlargement. If the lesion be below this level, that is to say, if it affect the sacral segments or cauda equina, no degeneration of the tract occurs; this fact is proved by several reported cases of such lesions. In other words, therefore, there is a short stretch of the spinal cord comprising these segments, situated between the 10th dorsal and the last lumbar segment, a lesion of which will cause ascending degeneration of the ascending antero-lateral tract, leaving the direct cerebellar tract intact. And it was from observation of such a case (one of crushing at the 11th dorsal level), that Gowers was enabled to describe the antero-lateral ascending tract as one quite distinct from the direct cerebellar tract, two tracts which had been formerly regarded as one. Bechterew independently confirmed this distinction by adducing developmental proof that

the fibres of this tract become myelinated at the eighth month of fœtal life, that is, later than the fibres of the "direct cerebellar tract."

The tract once formed takes up, according to Tooth, the following position: "it lies in the anterior part of the lateral column, in continuity in many cases with the direct cerebellar tract, behind and close to the anterior rootzone in front. It is not a thin strip like the direct cerebellar tract, but a wedge-shaped area, with its base at the periphery of the section, and its apex towards the grey matter and sometimes bent posteriorly, so as to insinuate itself between the crossed pyramidal and the direct cerebellar tracts. It is situated in an area of the cord called by Flechsig the "mixed zone." This position it more or less maintains throughout the cord.

Until some experiments, performed by Mott with a view to determining the destination of the antero-lateral ascending tracts were published, in 1892, great doubt existed concerning the upper course of this tract, though many, following Bechterew's lead, accepted the nucleus lateralis in the medulla oblongata as its terminus. Mott, by a novel experiment on the monkey, that of section of the fibres of the tract in the upper cervical region, by a specially devised instrument, and by the employment of the method of Marchi, was enabled to trace with the utmost accuracy the line of degeneration marking out the bulbar course of the tract. Tooth, in a subsequent paper, amply confirmed Mott's observations. Schäfer and Loewenthal independently arrived at similar results.

The following are the main anatomical points noted by these observers:—At the level of the nucleus lateralis the tract is seen at the periphery external to that nucleus and to the "so-called" ascending root of the fifth nerve. Dorsally, the tract touches the direct cerebellar tract; also many degenerated fibres are visible in the nucleus lateralis itself, thus a connection between the tract and that nucleus is proved, but it does not end there. A short distance higher up the antero-lateral ascending and direct cerebellar tracts separate from one another, and the divergence becomes wider as one ascends. At the level of the olive the ascending antero-lateral tract lies among the external arciform fibres dorsal to that nucleus. In a transverse section catching the sixth and seventh nerves in their intra-medullary course, the tract is still seen at the periphery between the point of exit of these nerves; but also dorsally in the angle formed by the brachium conjunctivum with the lingula, a small mass of degenerated fibres cut transversely becomes visible, and these represent the descending turn of a

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loop formed higher up by the passage of the ventral fibres of the tract over and round the root of the fifth cranial nerve. In sections made higher up the two portions of the loop approximate to one another, and at the level of the lowest point of exit of the fourth nerve the extremity of the loop can be discerned as a strip of fibres lying externally to the lateral lemniscus. Having looped in this manner the tract proceeds to the white centre of the superior vermis by way of the superior cerebellar peduncle and valve of Vieussens.

LITERATURE ON THE ANTERO-LATERAL ASCENDING TRACT.

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A Long Sensory Tract in the Grey Substance of the Cord—(Ciagliński).

In the *Neurologisches Centralblatt* for September, 1896, Adam Ciagliński draws attention to the existence of a long tract situated in and coursing along the grey matter of the cord,

and although his observation still requires confirmation, it is of such an interesting nature as to demand separate consideration.

Ciagliński drew a tight ligature round the cords of dogs in the lumbar region and killed the animals after from 4-24 hours. The cords examined by the method of Marchi showed, in addition to the usual ascending changes in the posterior columns, a diamond-shaped or rhomboidal area of degeneration situated in the grey commissure; between the ventral extremity of the posterior columns and the central canal, the area was composed of an actual column of fibres which, diminishing in volume as it ascended, was traceable upwards as far as the cervical enlargement.

This writer claims to have seen the tract in the normal human cord, but by this he can only mean a few sparsely disseminated fibres to be found in this region. He believes that its fibres are derived from the posterior roots and physiologically belong to that group of sensory fibres intended for the conveyance of impressions of heat and pain. If this be correct, and it is not an unsupported presumption, having been alluded to by Bellingeri in 1823, and Schiff in 1856, degeneration of the tract may explain certain sensory anomalies (loss of sensations of heat and pain) peculiar to syringomyelia.

LITERATURE.

CIAGLIŃSKI. "Lange sensible Bahnen in der grauen Substanz des Rückenmarkes und ihre experimentelle Degeneration." *Neurolog. Centralb.*, 1896, No. 17.

LATERAL LIMITING LAYER.

The lateral limiting layer, as described by Flechsig, is a part of the lateral columns contiguous or adjacent to the grey substance, which in the upper cervical region bounds practically the whole outer side of both cornua, in the cervical enlargement occupies the angle between the anterior and posterior cornua, and in the dorsal region again reaches forwards outside the anterior cornu, but altogether forms a less voluminous bundle than in the upper cervical region. In the lumbar region the layer requires further study.

Flechsig supposed that the layer was made up of fibres derived from two sources, the posterior roots and the cells of Clarke's columns, but we now know that this is incorrect, for, firstly, the layer does not degenerate in tabes dorsalis, nor does section of the posterior roots affect it, therefore it can have no direct con-

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nection with the posterior roots; and, secondly, it is believed that Flechsig mistook for fibres of this layer the neuraxons of the cells of Clarke's column which pass through the lateral limiting layer on their way to the direct cerebellar tract, as described by Mott. It is more likely, as Ramon y Cajal contends, that the layer is composed of fibres which, arising from cells in the anterior or posterior cornua, after a short course either upwards or downwards, again return to the grey matter.

In 1893 Bechterew drew attention to a special bundle of fibres in the posterior portion of Flechsig's lateral limiting layer, which he designated the "mediales Seitenstrangsbündel," he states that this bundle is distinguishable from Flechsig's layer in that its fibres develop their medullated sheath at a later period. It first becomes evident in the dorsal region situated immediately to the outer side of the grey substance between the lateral and posterior cornua, so occupying a portion of the territory between the lateral pyramidal tract and the cornua; further upwards the bundle advances more to the front, but still remains close to the grey substance. Bechterew, though doubtful concerning its constitution, presumes that its fibres stand in relation to cells in the grey substance, and that these fibres must be of very limited length, as they have never been seen degenerated in the human cord.

In a case of amyotrophic lateral sclerosis recently recorded by Bruce, the interesting point was determined that the anterior or ventral part of the lateral limiting layer had undergone partial sclerosis, while the posterior portion was preserved intact, and since atrophy of the anterior cornual cells is a fundamental change in amyotrophic lateral sclerosis, this observation in a measure proves that the anterior portion of the lateral limiting layer is connected with anterior cornual cells, and it suggests a relation between posterior cornual cells and the posterior portion. The layer may, therefore, be said to be composed of two independent systems which merely agree in that the fibres arise from cells in the grey matter and are of short course. Bruce suggests the names antero-lateral and dorso-lateral for these two divisions, but at the time that he wrote seems to have been unaware of the fact that Bechterew had already given a special description of the posterior division.

LITERATURE ON THE LATERAL LIMITING LAYER.

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BRUCE. "On a special tract in the lateral limiting layer of the spinal cord." *The Scottish Med. and Surg. Journ.*, vol. i., No. 1, 1897.

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ANTERIOR GROUND BUNDLE OR BASIS BUNDLE.

Excluding the fibres of the direct pyramidal tract, the antero-lateral ascending tract, the crossed afferent tract of Edinger (*vide* antero-lateral ascending tract), and the ascending and descending sulco-marginal tracts, the anterior column is composed of the anterior ground fibres, all of which lie in the immediate neighbourhood of the anterior cornu. The volume and the depth of the bundle varies at different levels. From the degenerative point of view it possesses little interest, as secondary changes in its fibres, even in a case of transverse myelitis, cannot be traced beyond the "traumatic zone." Flechsig and Bechterew, however, in their developmental researches, brought out several important points concerning these fibres; in the first place it was discovered that they received their medullary investment at an early date, viz., the fifth month (considerably earlier than the antero-lateral ascending tract, but not long before the direct cerebellar tract), then it was found that all do not become medullated at once, the first to develop being those which lie in immediate contact with the anterior cornu, and those are supposed to be related to fibres of the anterior roots; others which develop later, it is presumed, are short internuncial vertical fibres connecting cells in different levels of the anterior cornu.

CORNUO-COMMISSURAL BUNDLE (MARIE).

It would appear from experimental anatomical evidence that a bundle of fibres called by Marie the "faisceau cornu commissural," situated in that part of the column of Burdach which borders the posterior commissure, and also about the ventral half of the posterior cornu, cannot be grouped with the long fibres of the posterior columns, but must be classed among the short internuncial or anastomosing fibres connecting different levels of the grey substance. This bundle escapes degeneration after root lesions, and topographically corresponds with Flechsig's anterior

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rootzone or "ventrales Hinterstrangsfeld," with the exception that, according to Marie's representation, it is hardly so extensive. The tract requires further study.[1]

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CHANGES IN THE SPINAL CORD FOLLOWING AMPUTATION OF EXTREMITIES.

Since the early part of the century, contributions to the literature on this subject have been constantly appearing, and on glancing through the records one finds that while there have been minor differences among the various writers the general results obtained have been tolerably uniform. The predominant alteration is an atrophy of that half of the spinal cord corresponding to the side of the amputation. This has almost invariably been noticed, and is represented by a general reduction in volume of white and grey matter alike, involving those particular segments of the cord which receive and give off the sensory and motor nerves which originally supplied the skin and muscles of the amputated member or part thereof. That the atrophy is approximately confined to these particular segments may be definitely proved, if in a case of amputation these segments be carefully examined and compared with those above and below the suspected level; this was done in two cases coming under my notice, and the results were very definite. That the spinal hemiatrophy has not been definitely localised in this manner by previous writers is obviously owing to the fact that the areas appertaining to the various posterior spinal nerve roots have only been mapped out quite recently; but on analysing records and critically examining published drawings, one finds that the hemiatrophy has almost without exception been noted as being most prominent at the levels which I have emphasised. The cord above and below these levels gradually reassumes its symmetry, and the atrophy is of course not absolutely confined to them.

Studying these changes in greater detail, and in the first place taking the white matter, one finds that though an actual localised

[1] Since the above was written an article by Alexander Bruce "On the Endogenous or Intrinsic Fibres in the Lumbo-sacral Region of the Cord" has appeared in this Journal (part iii., 1897), and therein will be found an excellent description of this set of fibres.

sclerosis may occur, such a change is rare. Marie has reported two such cases, one of amputation of the thigh, another of amputation of the arm, and in both there was ascending bilateral sclerosis in the root area of the posterior columns, most marked on the side of the amputation. Two Italian observers, Guarneri and Bignami, also describe sclerosis of the antero-lateral ascending tract on the opposite side in consequence of an amputation. It appears that for the production of this sclerosis the amputation must be very old standing (in Marie's cases the operation had been performed twenty years previously, and Guarneri and Bignami's bore a similar history). Other changes which are constantly seen in the atrophied white matter are an increase in the nuclei of the neurilemma and an apparent proliferation of the glial elements, but it is worthy of note that though all the white columns of the cord on the side of the amputation may be appreciably reduced in volume, still their staining reaction in all the ordinary methods is practically identical with that of the healthy half.

Next, in regard to the grey substance, it has been mentioned that there is always a concomitant general reduction in its volume, likewise there is a general reduction in number of its contained nerve cells, both large and small, and furthermore there is one special group of cells which, both in the case of amputations of the upper and lower extremities, may be singled out as being particularly prone to atrophy, viz., the postero-lateral group of the anterior cornu. Grigoriew, who is the most recent writer on this subject, is disposed to dissent from this statement, but there is little question concerning its accuracy, for on analysing the literature, one is at once struck with the frequency with which the group is figured or described as having suffered most. Sherrington made a special note of this point in his research on the lumbo-sacral plexus, and uses it in support of his suggestion that in the lumbo-sacral region the postero-lateral group of nerve cells stands related to the intrinsic muscles of the foot. Yet another group of nerve cells which undergoes atrophy after an amputation of the lower extremity is that of the posterior vesicular column of Clarke. Friedländer and Krause first drew attention to this fact, and I noticed it in a case in which I stained my specimens by the method of Nissl.

From the experimental side the subject of the spinal changes after amputations has been vigorously investigated by Homén, and less seriously by Sherrington and others. The former, using the dog as his subject, has succeeded in reproducing most of the changes which have been observed in man; he mentions that

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young animals are most suitable for these experiments, and states that in puppies changes commence at the periphery somewhat more than a week after the operation, but in full-grown animals do not appear until one or two months have elapsed.

Friedländer and Krause and Erlitzky maintain that only the sensory neurons atrophy after amputations. Homén's experiments lead him to a similar conclusion, and he publishes apparently convincing coloured plates representing transverse sections of the posterior root ganglia, and showing plainly an intact motor root lying alongside the withered ganglion. But the integrity of the spino-muscular neuron here referred to, or even of the cortico-spinal neuron, can surely only apply to cases of more or less recent amputations, for there is abundant evidence on record to show that not only do the motor roots in course of time atrophy, but also motor anterior cornual cells, and even, likewise, cells in the motor sphere of the cerebral cortex (Charcot and Pitres, Wiglesworth, &c.).

Into the condition of the peripheral nerves in cases of amputation I do not propose to enter at length. The majority of observers state that they are reduced in calibre (Marie has seen them enlarged), the fibro-cellular tissue in them is increased in quantity, the perineurium is thickened, and sometimes myxoid or fatty, and the healthy fibres are reduced in number by about one-half; these changes diminish in severity as one ascends the nerve trunk.

Marie, who in his lectures refers in some detail to the alterations in the peripheral nerves, describes the presence in them of numerous islets, about four times the size of a healthy nerve-fibre, composed of a collection of some five to twenty delicate nerve fibres, possessing an almost imperceptible white sheath. These islets he looks upon as degenerated remnants of an originally healthy fibre, and calls *ilôts de dégénération*.

Some writers have described the posterior root ganglia as intact; others have found them diminished in size, their nerve-cells reduced in number, and their nerve-fibre plexus attenuated. We may take it that in old-standing cases of amputation they are always altered in the same way as the anterior and posterior spinal roots are.

What the precise pathology of the changes in the central nervous system after amputations is, is still *sub judice*. Clearly the change in the sensory nerve distal to the ganglion must be a true secondary degeneration, and we might expect atrophy of the sensory neuron above the ganglion in consequence of loss of function. The atrophy of the system of motor neurons may be

of the nature of a retrograde degeneration or sclerosis, similar to that described elsewhere in the cord. Obviously the spinal changes are not the result of a true secondary degeneration, and the distinguishing term "Gudden's atrophy" has been applied to them.

LITERATURE ON CHANGES IN SPINAL CORD AFTER AMPUTATIONS.

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WIGLESWORTH. "A case in which an old amputation of the left upper arm was associated with an atrophied right ascending parietal convolution." *Journ. of Ment. Science*, 1886.

SHERRINGTON. "Notes on the arrangement of some motor fibres in the lumbo-sacral plexus." *Journ. of Physiology*, vol. xiii., 1892.

Full lists of writers on this subject will be found in the papers of Sherrington, Homén and Grigoriew.

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HEREDITARY FORM OF PROGRESSIVE MUSCULAR ATROPHY WITH SPINAL LESION IN YOUNG CHILDREN.

(1) "Zwei Frühinfantile Hereditäre Fälle von Progresver Muskelatrophie unter dem Bilde der Dystrophie, aber auf Neurotischer Grundlage." Werdnig. (*Archiv für Psychiatrie*, 1891.)

(2) "Ueber Chronische Spinal Muskelatrophie in Kindesalter auf familliärer Basis." Hoffman. (*Deutsche Zeitschrift für Nervenheilkunde*, 1893.)

(3) "A Case of Progressive Muscular Atrophy in a Child with a Spinal Lesion." Thomson and Bruce. (*Edinburgh Hospital Reports*, 1893.)

(4) "Die Frühinfantile Progressive Spinale Amyotrophie." Werdnig. (*Archiv für Psychiatrie*, 1894.)

(5) "Weiterer Beitrag zur Lehre von der Hereditären Progressiven Spinalen Muskelatrophie in Kindesalter." Hoffmann. (*Deutsche Zeitschrift für Nervenheilkunde*, 1897.)

The above papers deal with a form of muscular atrophy occurring in children which as yet has not obtained general recognition.

The first paper, by Werdnig, deals with two cases, the former of which occurred in a boy, the eldest child of healthy parents. At the tenth month of life weakness of the legs was first noticed (the child having been previously in good health); this was followed by weakness of the muscles of the back, loss of power in the arms so that the child could no longer raise the hand to the mouth, this loss of power being preceded by shaking movements of the arms. The child could no longer support the weight of the head (which, however, was somewhat hydrocephalic). Sensibility was unimpaired; skin reflex was present, but the patella, biceps, and triceps reflexes were lost.

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The *post-mortem* revealed a condition of chronic hydrocephalus, some degeneration of the pyramidal tracts, atrophy of the cells of the anterior horns, especially in the cervical and lumbar enlargements, and degeneration of the anterior roots. The gastrocnemius muscle was alone examined and showed simple atrophy.

The second case, brother of the above, was, like him, noticed to be weak when 10 months old; the weakness started in the legs and back; choreiform movements and fibrillary tremors of the muscles were present. The knee-jerks were lost, sensibility was normal. The reaction of degeneration was present in some of the muscles. He died at the age of 6 years. The *post-mortem* and microscopical conditions are described in Werdnig's second paper. The cervical and dorsal cord appear to the naked eye to be normal in appearance; in the lumbar region the distinction between the white and grey matter is not clearly defined; the anterior roots are thin in comparison to the posterior and somewhat darker in colour. The muscles were for the most part extremely atrophied, pale, and many of them fatty.

Microscopically some change was noted in the cells of the nuclei of the motor portion of the 5th and in the 7th. The motor cortex was normal. The pyramids were normal. The cord showed atrophy of the cells of the anterior horn in the cervical and lumbar region; large cells appear but with cloudy contents and indistinct nuclei. The cells of Clarke's column appear to be normal, as well as the cells of the lateral horn. The anterior roots show considerable change; there are fewer bundles and an increase of nuclei and of interstitial tissue; some of the axis cylinders were swollen, and by the Marchi method showed some degeneration. The cauda equina was in parts completely degenerate, while in other parts was normal in appearance. The peripheral nerves, both with Marchi and with Pal, showed considerable degeneration.

In the muscles the most frequent change was simple atrophy, next a homogeneous degeneration, and lastly a ("schollig") degeneration; no fatty degeneration could be found. Large round muscle fibres remained at certain points, and a variable amount of fat and connective tissue; the condition of the muscles is not one of simple atrophy, but is a mixed process.

The first paper by Hoffmann deals with the case of a child (a girl) who, apart from being unusually fat, was well till the ninth month of life; from that time on the child began to lose power, first of standing, then of being able to sit up or turn itself round in bed. The child was intelligent, learnt to speak and never

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had any fits. In the place of the fat the extremities and the buttocks became wasted; no atrophy of the face occurred, and the child had no difficulty in swallowing. The child became absolutely unable to lift the head from the pillow, but could roll it from side to side. No fibrillar tremors, no trophic or vaso-motor disturbance were present. Sensation in all the forms remained normal. Pseudo-hypertrophy was never present. Reaction of degeneration was present. The child died when 4 years old. The autopsy showed marked atrophy of the anterior roots of the spinal cord; the muscles were a pale yellow colour, the quadriceps being the most atrophied.

Microscopical examination showed the brain to be normal; extensive atrophy of the cells of the anterior horn was present, but some unaltered cells existed in this region; atrophy of the anterior roots, and also of some of the fibres of the cauda equina and the peripheral nerves.

The muscles had undergone most extreme atrophy, so that in certain specimens not a single normal fibre could be found; hypertrophied fibres were present, and these contained more nuclei than normal; muscle schläuche (spindles) are described.

The intra-muscular nerves contain a fair number of degenerated and degenerating fibres, and rarification of the medullary sheath of the nerve is also described.

No alteration was found in the diaphragm, the masseter, or the cardiac muscle.

The second case, brother of the above, was first seen when $2\frac{1}{4}$ years old. As with his sister he was apparently a normal child till 9 months old, and although he never learnt to stand or walk, he even lost the power of sitting up in the cradle. The loss of power was gradual and not attended with pain. The child learnt to speak and was quite intelligent; he swallowed without difficulty.

When examined the child was fat but without obvious signs of rickets; it was unable to lift up its head and the arms were atrophied, even the small muscles of the hand being affected. The most marked weakness was, however, in the gluteal muscles and the muscles of the thigh: flexion of the hip could not be performed. The knee jerks were absent. This child died when 5 years old, but no autopsy was allowed.

The third case occurred in a boy, first seen when 9 months old, having loss of power in the gluteal, thigh and back muscles. Reaction of degeneration was present. The knee jerks were absent. The small muscles of the hand were affected, but there

was no fibrillary tremor. The child died when 14 months old, but no autopsy was obtained.

The report of a fourth case is also given without autopsy.

In the second paper by Hoffmann he reports a case, the sister of the child described in case No. 3 of his first paper.

This child, born of healthy parents and in natural labour, was apparently healthy till the seventh month of life, when it began without known cause, or sign of acute or chronic disease, to have weakness in the hip and thigh muscles of both sides; this weakness gradually extended to the muscles of the back, the neck and shoulder; later in the course of the disease the upper arm, the lower arm, and the muscles of the hand on one side and the leg muscles of the other side became affected. There were no fibrillary tremors and the sensation and the sphincters were normal. The knee jerks were absent. No electrical examination of the muscles was made.

The mental development was not impaired, and the face, tongue, and deglutition muscles were normal. Death took place at 2½ years from secondary lung affection.

Microscopical Examination.—The brain, cerebellum, the cranial nuclei and nerves were normal. From the medulla downwards the nerve cells of the anterior horn are atrophied or have entirely disappeared; the nervous network in the anterior horn is less than normal, and the neuroglia is thicker than natural.

The direct pyramidal and the lateral columns are by the Weigert method somewhat paler than normal, and Goll's column is rather paler than Burdach.

The anterior roots are reduced in volume and degenerated; in cross section there appear small islands of structureless tissue; these are thought to be nerve bundles which have become converted into connective tissue; they are found only at the exit of the anterior roots, and not in the posterior roots or in the cauda equina. Whole bundles of the nerves in the cauda equina are found to be degenerated.

Changes were also found in the sciatic and peroneal nerve, and to a lesser degree in the ulnar and radial.

The muscle fibres are reduced in size, measuring about 5-10 μ; they are round in form and the striation is well preserved even in the very smallest. There is also a diminution in the number of nuclei in the muscle fibre; normally there are about $1\frac{3}{4}$ of a nucleus to a fibre, while in the present case there is only about $\frac{2}{5}$ to $\frac{3}{5}$ of a nucleus to a fibre. There is no fatty degeneration or vacuolation; the intermuscular nerves are somewhat degenerated. Muscle spindles are noted as being present.

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The paper by Bruce and Thomson deals with the case of a child, the daughter of healthy parents, there being two other children in the family: (1) a boy who has congenital talipes varus; and (2) a girl who is strong and healthy.

At birth and during the first twelve months of life the child was quite healthy. Teeth began to appear at the sixth month, and at the twelfth month she could walk round the room holding on to a chair. Soon after the beginning of the second year of life she became unable to walk, and at eighteen months she could not get on to her feet. At the twenty-eighth month she could still stand with support, and was able to sit up and feed herself; she could sing and talk loudly; her arms remained fairly strong. When 3 years old she began to have pains in the legs, generally referred to the knee; the pain recurred at intervals for four months, but the attacks did not last long. On examining the child when 3 years old the appearance of limpness was very noticeable; the child could sit on a chair when balanced, but was very easily upset. She was bright and intelligent; there was some twitching of the angles of the mouth, and sometimes of the corrugator supercilii, also of the orbicularis oris and the orbicularis palpebrarum. Sensation to touch, to heat and cold were well preserved, but to the faradic current the child seemed quite insensitive. Deglutition, respiration, micturition, defæcation, perfectly normal. Knee jerks were absent; there was no A.C. The weakness was general; there was no special weakness of any group of muscles. The paresis was least marked in the shoulders and arms, more in the neck, back and abdomen, and most in the loins, buttocks and lower limbs. No hypertrophy or enlargement of any muscle. Co-ordination perfect.

Electrical examination showed diminished irritability to the faradic current, more marked in the legs than in the arms. The galvanic irritability was almost minimal. Distinct R.D. was never found. The sensibility to the faradic current was entirely abolished; the coil could be pushed up to its utmost limit without the child wincing.

The child died when 6 years old.

Post-mortem the brain and cord appeared normal; the muscles were pale and some were fatty.

Microscopical examination of the muscles showed all degrees of atrophy; hypertrophied fibres were present, and an increase of the nuclei of the sarcolemma; the striation was well preserved in all except the smallest fibres. The occurrence of minute muscular fibres in the centre of a nucleated fibrous ring is noted.

sometimes these rings are divided into compartments, one of which contains a nerve fibre. These are muscle spindles.

The sciatic and nerves of the brachial plexus were examined by Weigert's method, and atrophy of some fibres and attenuation of the myelin sheath of other was found. The spinal cord showed no tract degeneration; the posterior roots were normal, but the anterior showed slight atrophy. The cells of the anterior horn had to a great extent disappeared, and those remaining had undergone considerable atrophy. In the lumbar region the process had proceeded so far that only a few highly atrophic cells could be seen. None of them presented any abnormal pigmentation.

There remains an uncertainty as to which was the primary lesion in this case. The authors, however, think that it might be classed under the "simple atrophic" form of Erb.

The characteristics of the disease, as illustrated by the above cases, are, then, as follows: an apparently healthy and intelligent child who has made normal progress to the age of 10 months, begins without any sudden onset or known cause to lose power, the weakness being first noticed in the muscles about the hips and in the muscles of the back. The disease pursues a progressive course, the shoulders, the thighs, the upper arm, the forearm and leg being successively involved, and finally the muscles of the hands and feet become affected, the parts being involved in the order above-mentioned. No special group of muscles are affected. Fibrillary twitchings of the muscles are present in some cases. Bulbar symptoms may supervene, and contractions of the limbs may be present in some cases (Werdnig). The limbs are absolutely flaccid, reaction of degeneration is often present, the deep and sometimes the superficial reflexes are abolished. There is as a rule no tenderness or pain, though the latter may be present (see Thomson and Bruce's case). There is no disturbance of sensation, but again in the case just referred to there was an extraordinary absence of pain to the stimulus by the faradic current. The sphincters are normal. The mental condition continues unimpaired throughout the whole course of the disease. The atrophy of the muscles becomes extreme, and the disease runs its course in at most a few years. Heredity plays an important part in the etiology of the disease; in one family of six, two brothers were affected; in another of fifteen, four boys and two girls were affected; in a third family of nine, two boys and a girl were affected; and in a fourth family of twelve, eight were affected in one generation, and two girls and a boy in the

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second generation, through the mother. The evidence on which some of the above is based is open to doubt, as out of the above twenty-two cases only four were examined pathologically.

In Thomson and Bruce's case there was no hereditary or family history. The pathological condition which has been found in these cases is atrophy of the cells of the anterior horn, together with degenerative changes in the anterior nerve roots and in the peripheral nerves. In the muscles sometimes simple atrophy is found, sometimes the condition usually found in cases of primary muscular atrophy.

Whether the atrophy in the cells of the anterior horns is the primary lesion or is secondary to the condition of muscular atrophy is a point that is difficult to decide; certain evidence in the above cases would seem to point to the probable spinal origin of the disease.

F. E. Batten.

Reviews and Abstracts.

Eye-Strain in Health and Disease; with special reference to the amelioration or cure of Chronic Nervous Derangement without the aid of Drugs. By AMBROSE L. RANNEY, A.M., M.D., 1 vol., 8vo., p. 321. Davis Co., Philadelphia.

IN this volume the author has brought together the substance of several monographs, published in past years in various medical journals, and has added considerable new matter.

It evinces signs of much careful and laborious work, and deserves, therefore, patient and impartial consideration on the part of the reader. The tone of the book as a whole is, perhaps, somewhat excessive and extravagant, but this may possibly be explained by the desire of a man, with an enthusiastic belief in his subject, to impress strongly his views of the case upon the world. Some fifty or sixty pages are given up to a very concise and remarkably clear account of the steps to be taken in examining the vision and the ocular movements. This, a little enlarged and improved, would make a useful text-book if published in a separate volume, but seems, to a certain extent, out of place in its present environment.

The so-called "Heterophoria" occupies a very important position in this work, one equal, if not superior, to that held by errors of refraction, with regard to the causation and treatment of nervous diseases. In this country we do not recognise it as being of very frequent occurrence, nor as producing such enormously injurious results to the nervous system. We acknowledge that lack of equilibrium of the ocular muscles exists, and have done so for many, many years, but we think that "heterophoria" and its treatment by operation (graduated tenotomy), as at present practised, is carried too far by some of our brethren in the United States. Possibly the citizens of the United States suffer from this trouble more than we do.

The rest of the book consists, in a large part, of cases of many and various kinds, showing the results of treatment by glasses and operation, interspersed with remarks and arguments. The great

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principle which Dr. Ranney advances strongly is the removal of peripheral irritation, and he desires to show that the eyes in a large number of cases are undoubted sources of trouble. He deprecates the indiscriminate use of drugs, especially the bromides, and earnestly begs for the careful examination of the eyes and other organs of the body before medicines are employed. All this is just and proper, and cannot be too frequently set forth before the profession at large. Dr. Ranney recognises neurasthenia, &c., as a frequent result of eye-strain, but, curiously enough, says little about cases of eye-strain as a consequence of nervous trouble, which latter form a large portion of the patients who have to be treated for eye trouble.

The book is worth reading. In doing so, however, it is necessary to guard oneself against an impression produced, viz.: that eye-treatment cures or relieves everything.

H. Work Dodd, F.R.C.S.

The New Psychology. By E. W. Scripture, Ph.D. (Contemporary Science Series. Walter Scott, London.)

This is distinctly a good book. Although laying no claim to to be an exhaustive compendium like those of Wundt, Ladd, and Külpe, it is in reality a great deal more compendious, as well as more readable, than most other books of equal size. To anyone —whether physiologist or neurologist—who has had a passing acquaintance with the literature of experimental psychology of recent date, it is a valuable *résumé* and digest of some of the best work that has appeared. To the professing laboratory psychologist it is valuable by reason of the numerous experimental hints and the considerable amount of original matter embodied in its pages.

Part I., consisting of four chapters on Observation, Statistics, Measurement, and Experimenting, are couched in a tone that should go far to allay the suspicions with which most scientific readers are apt to view an exponent of the "new psychology." The figure on p. 65, from Hansen and Lehmann, serves as a rather startling illustration of the way in which one successfully makes out points of resemblance in apparent "proof" of thought-transference. The similarity in this case is at first sight close enough to arouse the incredulity of an ordinary observer, or to

satisfy a psychical researcher that thought has been "transferred," yet it is clear enough on closer investigation that the apparent similarities are mainly invented by the observer, *i.e.*, oneself. The contrast between this investigation and that of an untrained scientist (a well-known psychical "researcher") is very effectively brought out by the brief summary of the "experiments" on thought-transference by Ochorowicz.

Part II. consists of an account of measurements of various kinds of reaction-times, and of the time-factor in memory, and of fatigue-phenomena. Several useful pieces of apparatus are figured and described; and the spark-dot method of recording long series of reaction-times strikes one as bringing out results in a very elegant form. Exner's sensation-curves, and the plotted data upon which the logarithmic law of declining memory is based, catch the eye as the pages are turned over, and are welcome. But the internal mechanism of the kinetoscope is not particularly welcome—in this connection at least.

Part III. deals with the manifestations of energy, and with the sensational effects of stimulation. Dynamometry, fatigue, the influence of mental states, and of various kinds of sensificatory stimuli upon volitional power are considered; the plotted curves exhibiting the effects of cerebral fatigue upon the movements of the eyes and upon the speed of accommodation are of special interest; these, as well as the "tapping-frequency" experiments mentioned in an earlier chapter, are the results of work done by the author or under his supervision. This is also the case in the measurements of suggestibility described at pp. 272-282. The tricolour lantern figured on p. 349, appears to be an excellent means of demonstrating colour mixtures on the screen.

Part IV. is concerned with the psychological dimensions of space.

Part V., of which one chapter is devoted to a biography of Fechner, Helmholtz and Wundt, takes stock of the present state and immediate future of the new or quantitative psychology. The author takes a sober and temperate view of its scientific gravity in the present, but a very hopeful view indeed of its development and ultimate fruitfulness.

Several useful tables are contained in the Appendix, and the following note (p. 484) on the measurement of illusions in it strikes one as capital. One does not feel the title to be hopeful, nor yet the opening paragraph, but the two concluding ones are quite satisfactory and eminently convincing of the soundness and ingenuity of their author. The paragraph runs thus:

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"*On the Measurement of Imagination.*—In the course of the investigations on measuring hallucinations it occurred to me that it might be possible to measure the intensity of an imagination also. The experiment was successful. The method is not difficult and is readily intelligible. In order to explain the method it will be sufficient to describe the first simple experiment made.

"The apparatus consists of a screen of fine tissue paper. The tissue paper is illuminated by daylight in front and by a gas flame at the back. When the gas flame is turned down, the eye looking through a telescope tube sees a plain white circle illuminated by daylight.

"The first experiment was made on a student accustomed to using the telescope. He was told to imagine hair lines on the white surface, like the hair lines seen in an astronomical telescope. This was successfully done. He was asked to describe them and compare their blackness. There is, he said, a horizontal line, which is the blackest of them, and three vertical lines of about equal blackness. He was told that the field of view was to be made gradually lighter by turning on a flame behind, and he was to tell how the lines behaved. As the gas was slowly turned on, he described various changes in the lines. Finally he said he saw a slant line that he had not imagined before. It appeared just about as black as the horizontal line and blacker than any of the others. Thereupon the experiment was ended.

"The slant line was a real line. This he did not and still today does not know. On the back of the tissue paper a slant line had been drawn, and as the gas was turned up of course it showed through. Thus we had a direct, unsuspecting comparison of intensity between a real line and an imaginary one.

"The photometric determination of the intensity of the real line is not a difficult matter. A phantasimeter has been devised in which the graduation is done beforehand, but the simple arrangement just described serves to indicate the method of experiment."

A. D. W.

Anæsthesia of the Trunk in Locomotor Ataxia. By Hugh T. Patrick, M.D., *New York Medical Journal*, February 6, 1897.

Sensory Disturbances in Locomotor Ataxia; a Study of the Localisation of Anæsthetic Areas as an Early Symptom. By Allan Blair Bonar, M.D., *Medical Record*, New York, May 22, 1897.

Hitzig and Laehr first drew attention to the sensory affections of locomotor ataxy. Patrick and Bonar have carefully investigated twenty and twenty-one cases of tabes respectively, with special reference to the areas of sensory disturbance on the trunk.

Seventeen of Patrick's cases and eighteen of Bonar's exhibited such areas. Laehr had found them in fifty-five out of sixty cases.

The sensory disturbances in question are mostly in the form of tactile anæsthesia, but there may be analgesia, and in a few cases loss of sensibility to heat and cold. They occur as more or less broad bands in the neighbourhood of the nipple. They may be unilateral or bilateral, with corresponding areas on the back. They may extend to the mid line, or fall short of it. Sometimes there are intervening areas of normal sensation between plaques of tactile anæsthesia. When bilateral the areas may be at different levels. Commonly above the anæsthetic areas there is a zone of hyperæsthesia, mostly (according to Hitzig) to cold.

When the anæsthesia reaches the level of the third rib it also involves a tongue-shaped portion of the axillary side of the corresponding arm, and when it extends above this level in the chest the anæsthesia spreads throughout the distribution of the ulnar nerve.

The anæsthesia does not correspond to the distribution of the intercostal nerves, but answers to the segments of the spinal cord, as differentiated by Kocher.

This is, however, not usually the case, Bonar says, when the sensory disturbance affects the lower extremities.

Laehr states that anæsthesia of the trunk only extends downwards, but both Patrick and Bonar have found numerous exceptions to this rule.

All agree that the anæsthesia is extremely variable, and that its boundaries are more easily defined when approached from within their limits than from outside.

Anæsthetic areas may increase or decrease, and even disappear, or hyperæsthesia may give place to anæsthesia. The conditions do not advance progressively with the course of the

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disease. They may be found in cases of 3 to 5 years' duration, and may be hardly marked in those which have lasted 25 to 28 years.

They are usually least apparent in cases where optic atrophy is prominent. The superficial reflexes are increased over hyperæsthetic zones, and diminished or absent in those of anæsthesia.

As regards the causation of tactile anæsthesia of the trunk, Patrick points out that any process which involves the posterior nerve roots, such as tumour, meningitis or spinal caries, may produce it; it may be present in syringomyelia and hysteria. In tabes he regards it as evidence of intra-medullary affection of the long fibres, which pass directly upwards in the posterior columns, leaving uninjured those that pass by various ways into the posterior grey horns and columns of Clarke.

As regards the diagnostic value of trunk anæsthesia, Patrick thinks it is probably not very great.

It seemed of value only in one case, and it was by no means absolutely certain that this was a case of tabes.

Laehr found it early, but except in one case not so early as analgesia of the legs.

Bonar regards paræsthesiæ in general as valuable evidence of early tabes.

He found them in a number of consecutive cases, and says that they often exist unknown to the patient unless involving the hands, feet, or buttocks.

The present writer has usually found some amount of tactile anæsthesia of the trunk in well-marked cases of tabes, but not in doubtful cases. One patient for upwards of a year has had progressive atrophy of both discs—Argyll Robertson phenomenon—and extremely sluggish knee jerks; but his sensation is perfect from head to foot. This, however, perhaps bears out Laehr's statement that sensory disturbances are commonly absent when optic atrophy is prominent.

LEONARD G. GUTHRIE.

PROCEEDINGS OF THE SOCIETY.

At a Meeting of the Society held on December 8th, Dr. Hughlings Jackson delivered the first Hughlings Jackson Lecture, the subject of which was "Remarks on the Relations of Different Divisions of the Central Nervous System to one another and to Parts of the Body."

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Rules

AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London,

1898,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1897.

LONDON:
JOHN BALE, SONS & DANIELSSON, LTD.,
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1898.

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Neurological Society of London.

REPORT OF COUNCIL.

JANUARY 13, 1898.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The Members now number 187.

The Inaugural Address was delivered at the Annual General Meeting on January 14th, 1897, by Dr. G. H. SAVAGE, on "Heredity in the Neuroses."

A special feature in the year's programme has been the delivery of the first Hughlings Jackson Lecture on December 8th, 1897, by Dr. HUGHLINGS JACKSON himself, in whose honour the Lectureship was founded by the Society last year. The subject of the Lecture was "Remarks on the Relations of Different Divisions of the Central Nervous System to one another and to Parts of the Body."

A satisfactory arrangement has been made with Messrs. MACMILLAN & Co. in view of the increased cost of publishing *Brain*, as they have undertaken to hand over to the Society one-third of their share of the net profits from the sale of the Journal, as a contribution towards the cost of illustrations.

The Council regrets the loss sustained by the Society by the death of an illustrious Honorary Member, Professor Du Bois Reymond, of Berlin; also of Dr. JAMES CAGNEY and Dr. E. FRICKE.

The Proceedings have been published at greater length in *Brain* than in former years, and an abstract appended to this Report shows the character and value of the work of the Society during the past year.

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Proceedings of the Society during the year 1897.

January 14th.—Dr. G. H. SAVAGE, Inaugural Address, "Heredity in the Neuroses," *Brain*, vol. xx., p. 1.

March 4th.—Dr. F. E. BATTEN made a communication on "The Muscle Spindle under Pathological Conditions," and illustrated his paper by lantern slides and by specimens under the microscope. Professor SHERRINGTON and Mr. VICTOR HORSLEY also made communications on the Muscle Spindle, and illustrated their remarks in the same way.

April 22nd.—Dr. FERRIER, "Two cases of Myoclonus Epilepticus." Dr. DONKIN (1) "Probably Subacute Poliomyelitis in a man aged 45 years"; (2) "Tabes Dorsalis with a great Preponderance of Sensory Disturbance"; (3) "Pseudo-hypertrophic Paralysis in an Advanced Stage." Dr. GOSSAGE, "Progressive Muscular Atrophy Commencing at the Early Age of 10½ years." Dr. LUNN, Dr. BEEVOR, and Mr. BALLANCE, "Case of Removal of a Cerebellar Tumour, two years and five months after the operation." Dr. JAMES TAYLOR, "Syringomyelia with Bulbar Symptoms." Dr. Harris, "Syringomyelia with Supposed Hæmorrhage into the Cavity."

June 3rd.—Professor SCHÄFER, "Is there a Direct Relationship between the Motor Paralysis produced by Lesions of the Cortex and Loss of Sensibility, Muscular or other?" The paper was illustrated by Lantern Slides, three living monkeys and the brain of a fourth that had been killed. Professor BOYCE and Dr. WARRINGTON, "Contributions to the Anatomy of some of the Ascending and Descending Nerve Tracts in the Fowl." (Lantern Demonstration.)

October 28th.—Dr. J. A. ORMEROD on behalf of Drs. SANGER BROWN and ADOLF MEYER. "Morbid Anatomy in a case of Hereditary Ataxy of the type described by Sanger Brown." (Illustrated by specimens under the microscope.) Drs. SAMUEL GEE and H. H. TOOTH, "Case of Hæmorrhage in the Region of the Pons Varolii, almost entirely on the Right Side." (Lantern Demonstration.) Dr. W. A. TURNER, "Symmetrical Softening of the Pyramids and Inter-olivary Strata." (Lantern Demonstration.) Dr. JAMES TAYLOR and Mr. C. A. BALLANCE, "Case of Tumour of the Right Lateral Lobe of the Cerebellum." Dr. RISIEN RUSSELL, "Degenerations in the Spinal Cord after Removal of a Large Tumour from one Cerebral Hemisphere, and in the case of a Tumour of the Lumbo-Sacral Cord."

December 8th.—Dr. HUGHLINGS JACKSON delivered the first Hughlings Jackson Lecture, the subject of which was "Remarks on the Relations of Different Divisions of the Central Nervous System to one another and to Parts of the Body."

BALANCE SHEET, 1897.

NEUROLOGICAL SOCIETY.

RECEIPTS.

	£ s. d.	£ s. d.
To Balance—		
General Balance ...	41 6 11	
Sir W. Broadbent ...	8 8 0	
		49 14 11
Subscriptions for current year		171 4 0
Do. in advance		1 0 0
Do. in arrears		12 0 0
Share of profit on "Brain"		46 10 4
Contribution by Messrs. Macmillan towards cost of illustrations in "Brain"		15 0 0
		£295 9 3

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EXPENDITURE.

	£ s. d.	£ s. d.
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Pulman & Sons	7 15 3	
Bale & Danielsson ...	9 9 9	
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Bale & Danielsson ...	75 11 6	
Pearson	2 0 0	
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Fielder (Univ. Coll.) ...	1 7 6	
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Balance		46 5 6
		£295 9 3

Audited and found correct, { J. B. LAWFORD.
December 17th, 1897. { W. S. COLMAN.

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

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11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—Absence of any Member residing within the Metropolitan area from all Meetings held during the year shall be considered as equivalent to resignation.

16.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

17.—Five Members of the Council shall form a quorum.

18.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

19.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

20.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

21.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

22.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

23.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

24.—There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

25.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

26.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty members.

27.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

28.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

29.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

30.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

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„ Munk, Berlin.
„ Obersteiner, Vienna.
„ Retzius, Gustaf, Stockholm.
„ Ribot, Paris.
Dr. Weir Mitchell, Philadelphia.

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1897 George H. Savage, M.D.

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*** Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.P., Vice-President.	C., Member of Council.

H.J.L., Hughiings Jackson Lecturer.

ORDINARY MEMBERS.

Elected.

1892 ALEXANDER, ROBERT REID, M.D., The Asylum, Hanwell, W.

1897 ANDERSON, H. KERR, M.B., B.Sc., 9, Park Terrace, Cambridge.

1894 ANDRIEZEN, W. LLOYD, M.D., Darenth Asylum.

1891 BALLANCE, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897.)

O.M. BARLOW, THOMAS, M.D., F.R.C.P., 10, Wimpole Street, W.

,, BASTIAN, H. CHARLTON, M.D., F.R.C.P., F.R.S., 8A, Manchester Square, W. (Pres., 1892; V.P., 1891; C., 1886-9, 1892-5.)

1897 BATTEN, FREDERICK E., M.D., M.R.C.P., 124, Harley Street.

,, BEACH, FLETCHER, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

,, BEEVOR, C. E., M.D., F.R.C.P., 33, Harley St., W. (Tr., 1894; C. 1893.)

1888 BIRT, ERNEST, M.D., L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.

1898 BOLTON, JOSEPH SHAW, M.D., B.S., B.Sc., Mason College, Birmingham.

1896 BOND, C. HUBERT, M.D., B.Sc.Edin., London County Asylum, Banstead.

1892 BOWLBY, ANTHONY A., F.R.C.S., 24, Manchester Square, W.

1889 BRADFORD, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 60, Wimpole St., W.

O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894.)

1895 BRAMWELL, JOHN MILNE, M.B., C.M., 2, Henrietta Street, W.

1892 BRISTOWE, HUBERT CARPENTER, M.D., L.R.C.P., M.R.C.S., 18, Warwick Road, Maida Vale.

O.M. BROADBENT, Sir WM. H., Bart., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.P., 1893; C., 1886-7, 1895.)

1894 BRODIE, T. GREGOR, M.D., L.R.C.P., Lindfield, Uxbridge Road, Surbiton.

1897 BROWN, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 BROWN, SANGER, M.D., 34, Washington Street, Chicago.

O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed., 13, Alva Street, Edinburgh. (C., 1895.)

O.M. BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.

,, BRUNTON, T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896.)

XUM

1895 Bryan, Frederick, M.B.Dunelm, M.R.C.P., The Asylum, New Southgate, N.

1895 Bryant, John Henry, M.D.Lond., L.R.C.P., Guy's Hospital, S.E.

1890 Bury, Judson Sykes, M.D., M.R.C.P., 10, St. John Street, Manchester.

O.M. Buzzard, Thomas, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-9; C., 1886-7, 1891-5.)

1889 Carter, Robert Brudenell, F.R.C.S., 31, Harley Street, W.

1887 Cattell, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.

1891 Clarke, J. Michell, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol.

O.M. Cobbold, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.

1892 Cole, Robert Henry, M.D., M.R.C.P., M.R.C.S., 53, Upper Berkeley Street, Portman Square.

1890 Colman, W. S., M.D., F.R.C.P., 22, Wimpole Street, W.

,, Corner, Harry, M.D., L.R.C.P., Brook House, Southgate, Middlesex.

1894 Cuming, James, Prof., M.S., F.R.C.P.I., 33, Wellington Place, Belfast.

1895 Davidson, Andrew, M.B., C.M., The County Asylum, Dorchester, Dorset.

1897 Dawson, William R., M.D., B.Ch., Farnham House, Finglas, Co. Dublin, Ireland.

1894 Dean, H. Percy, M.D., F.R.C.S., 69, Harley Street, W.

O.M. Donkin, H. B., M.D., F.R.C.P., 108, Harley Street, W. (C., 1897.)

,, Dreschfeld, Julius, M.D., F.R.C.P., 325, Oxford Road, Manchester. (V.P. 1898.)

,, Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1889 Eccles, A. Simons, M.B., M.R.C.S., 23, Hertford Street, Mayfair, W.

O.M. Edmunds, Walter, M.B., F.R.C.S., 75, Lambeth Palace Road, S.E.

,, Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1894; V.P., 1892-3; C., 1886-90-95.)

,, Foster, Michael, M.D., F.R.S., Shelford, Cambridge.

1888 Fox, Arthur, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.

O.M. Fox, E. Long, M.D., F.R.C.P., Church House, Clifton, Gloucestershire.

,, Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (C., 1896; V.P., 1897.)

,, Gee, Samuel, J., M.D., F.R.C.P., 31, Upper Brook Street, W.

,, Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1890 Goodall, E. W., M.D., Eastern Hospitals, Homerton, E.

1897 Gossage, Alfred Milne, M.B., M.R.C.P., 54, Upper Berkeley Street.

1892 Gotch, Francis, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 Grant, Dundas, M.D., F.R.C.S., 8, Upper Wimpole Street, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W.

1895 Guthrie, Leonard, M.D., M.R.C.P., 15, Upper Berkeley Street, W.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., 9, Ridgemount Gardens, W.C.

XUM

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1897 Harris, Wilfred J., M.D., M.R.C.P., 7, Craven Mansions, Craven Terrace, W.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 56, Portland Place, W.

O.M. Haycraft, J. B., M.B., F.R.S.E., The University, Cardiff.

1891 Head, Henry, M.D., C.M., M.R.C.P., 61, Wimpole Street, W.

O.M. Hill, Alex., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.P., 1895; C., 1892-94.)

,, Hopkins, John, F.R.C.S., London Sick Asylum, Cleveland Street, W.

,, Horsley, Victor, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.P., 1896; C., 1892-3.)

1889 Huggard, William R., M.D., M.R.C.P., Davos Platz, Switzerland.

1896 Hulme, George Frederick, M.D., M.S.Ed., Montague Road, Felixstowe.

1889 Humphry, Laurence, M.D., M.R.C.P., 3, Trinity Street, Cambridge.

O.M. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)

1889 Hyslop, T. B., M.D., C.M., Bethlehem Royal Hospital, Lambeth, S.E.

O.M. Jackson, J. Hughlings, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887-92.)

,, Jessop, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 Johnston, George, M.D., M.R.C.P., 6, Manchester Square, W.

1892 Jones, Henry Lewis, M.D., F.R.C.P., 9, Upper Wimpole Street, W.

1894 Jones, Robert, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

,, Kidd, Percy, M.D., F.R.C.P., 60, Brook Street, W.

1896 Langdon, F. W., M.D., Cincinnati, Ohio, U.S.A.

O.M. Langley, J. N., M.A., D.Sc., F.R.S., Trinity College, Cambridge. (C., 1898.)

1888 Lawford, J. B., M.D., F.R.C.S., 99, Harley Street, W.

O.M. Lees, D. B., M.D., F.R.C.P., 22, Weymouth Street, W.

,, Lewis, H. Wolseley, M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

1897 Lowenthal, Max, M.D., M.R.C.P., 84, Princes' Road, Liverpool.

1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.

,, Mackenzie, Hector William Gavin, M.D., F.R.C.P., 59, Welbeck Street, W.

O.M. Mackenzie, Stephen, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896.)

1896 Mackintosh, Ashley W., M.A., M.D., Wellington House, Alford Place, Aberdeen.

1889 Macphail, S. R., M.D., Borough Asylum, Rowditch, Derby.

O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

,, Mann, J. Dixon, M.D., F.R.C.P., 16, St. John Street, Manchester.

1894 Martin, Sidney, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.

1893 Maude, Arthur, L.R.C.P., M.R.C.S., Westerham, Kent.

XUM

1891 MAY, W. PAGE, M.D., M.R.C.P., Helouan, Cairo, Egypt.

1890 MENZIES, WILLIAM FRANCIS, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.

O.M. MERCIER, C., M.B., F.R.C.S., Flower House, Southend, Catford, S.E.

MEYER, ADOLF, M.D., Clark University, Worcester, Mass., U.S.A.

,, MICKLE, W. JULIUS, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (C., 1895-97.)

1895 MOORE, NORMAN, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. MORRIS, MALCOLM, F.R.C.S.Ed., 8, Harley Street, W.

1888 MOTT, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894, 1897.)

1894 MURRAY, GEORGE, M.B., M.R.C.P., 2, Saville Place, Newcastle-on-Tyne.

1889 MURRAY, H. MONTAGUE, M.D., F.R.C.P., 27, Savile Row, W.

O.M. NETTLESHIP, EDWARD, F.R.C.S., 5, Wimpole Street, W.

,, NICOLSON, DAVID, C.B., M.D., M.R.C.P.Ed., Elmhyrst, Guildford, Surrey.

1888 NIERMEYER, J. H. H., M.D., Amsterdam.

,, NORMAN, CONOLLY, F.R.C.P.I., F.R.C.S.I., Richmond Asylum, Dublin.

1887 OGILVIE, GEORGE, M.B., M.R.C.P., 22, Welbeck Street, W.

,, OGILVIE, LESLIE, M.B., M.R.C.P., 46, Welbeck Street, W.

,, OLIVER, THOMAS, M.D., F.R.C.P., 7, Elison Place, Newcastle-on-Tyne.

O.M. ORANGE, WM., C.B., M.D., F.R.C.P., The Bryn, Godalming, Surrey.

,, ORD, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)

,, ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

,, PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (C., 1891-2.)

1895 PARKINSON, JOHN PORTER, M.D.Lond., M.R.C.P., 40, Wimpole Street, W.

1896 PATERSON, DONALD ROSE, M.D.Edin., M.R.C.P., 18, Windsor Place, Cardiff.

1895 PINDER, GEORGE, M.B., B.C., Seafield House, Ramsey, Isle of Man.

1888 PITT, G. N., M.D., F.R.C.P., 24, St. Thomas's Street, S.E.

O.M. POORE, G. VIVIAN, M.D., F.R.C.P., 32, Wimpole Street, W. (C., 1898.)

1887 PRINGLE, J. J., M.B., F.R.C.P., 23, Lower Seymour Street, Portman Square, W.

O.M. PURVES, W. LAIDLAW, M.D., 20, Stratford Place, W.

1894 RANSOM, WILLIAM B., M.D., M.R.C.P., The Pavement, Nottingham.

1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 2, Harley Street, W.

O.M. REID, E. W., M.B., University College, Dundee.

1897 REISSMANN, CHARLES H., B.A., B.Sc., Saxony Villas, Oppidans Road, Primrose Hill.

1893 RENNIE, GEORGE E., M.D., 16, College Street, Sydney, N. S. Wales.

1895 REYNOLDS, ERNEST S., M.D., F.R.C.P., 23, St. John Street, Manchester.

1892 RICHARDS, JOSEPH PEEKE, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 RIVERS, W. H. R., M.A., M.D., M.R.C.P., St. John's College, Cambridge. (C. 1897.)

XUM

1892 ROWE, EDMUND LEWIS, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

1892 RUFFER, MARC ARMAND, M.D., Cairo.

1889 RUSSELL, J. S. RISIEN, M.D., F.R.C.P., 4, Queen Anne Street, W. (Sec., 1896-1897.)

O.M. SANDERSON, J. BURDON, M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

,, SAVAGE, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres. 1897; V. P., 1895; C., 1886-90, 1898.)

1888 SAVILL, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. SCHAEFER, E. A., F.R.S., University College, Gower Street. (Pres., 1893; V.P., 1892; C., 1886-90.)

,, SEMON, Sir FELIX, M.D., F.R.C.P., 39, Wimpole Street, W.

,, SHARKEY, S. J., M.D., F.R.C.P., 22, Harley Street, W. (C., 1889-92.)

,, SHERRINGTON, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 SHUTTLEWORTH, GEORGE E., M.D., Ancaster House, Richmond.

1895 SMITH, F. J., M.D., M.R.C.P., 4, Christopher Street, Finsbury Square, E.C.

1887 SMITH, R. PERCY, M.D., F.R.C.P., Bethlem Royal Hospital, Lambeth.

1895 SMITH, TELFORD, M.D., B.S., Royal Albert Asylum, Lancaster.

1895 SPICER, WILLIAM T. H., M.D.Cantab., F.R.C.S., 47, Welbeck Street, W.

1895 STANSFIELD, THOS. E. K., M.B., Claybury Asylum, Woodford, Essex.

1895 STANLEY, DOUGLAS, M.D.Edin., 9, Easy Row, Birmingham.

1892 STARLING, ERNEST HENRY, M.D., F.R.C.P., B.S., 8, Park Square, N.W.

1897 STEWART, PURVES, M.A., M.D., M.R.C.P., National Hospital, Queen Square, W.C.

O.M. STEWART, Sir T. GRAINGER, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.

,, STIRLING, WM., M.D., D.Sc., Owens College, Manchester.

1897 STODDART, WM. H., M.B., B.S., M.R.C.P., County Asylum, Prestwich, Manchester.

1895 STOUT, G. F., M.A., St. John's College, Cambridge.

1887 SUCKLING, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

O.M. SULLY, JAMES, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P., 1890-91.)

1889 SUTHERLAND, HENRY, M.D., M.R.C.P., 21, New Cavendish Street, Portland Place, W.

1888 SYERS, HENRY W., M.D., M.R.C.P., 4, Oxford and Cambridge Mansions, Hyde Park, W.

1891 TAYLOR, JAMES, M.D., F.R.C.P., 49, Welbeck Street, W.

1898 THOMSON, H. CAMPBELL, M.D., M.R.C.P., 34, Queen Anne Street, W.

1889 THORBURN, WM., F.R.C.S., The Limes, Nelson Street, Manchester.

1892 TITCHENER, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.

O.M. TOOTH, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896.)

1892 TREVELYAN, E. F., M.D., B.Sc., M.R.C.P., 40, Park Square, Leeds.

XUM

1892 Tuckey, Charles Lloyd, M.D., C.M., 88, Park Street, Grosvenor Square, W.

1892 Tuke, Sir J. Batty, M.D., F.R.C.P.Ed., 20, Charlotte Sq., Edinburgh.

1891 Tuke, Thomas Seymour, M.B., M.R.C.S., Chiswick House, Chiswick.

1891 Turner, William Aldren, M.D., F.R.C.P., 13, Queen Anne Street, W.

O.M. Tweedy, John, F.R.C.S., 100, Harley Street, W.

1888 Voorthuis, J. A., M.D., Medan, Deli, East Coast of Sumatra. (Communications to be addressed to M. Seyffardt, Bookseller, Amsterdam.)

1895 Wade, Arthur L., M.D., Somerset and Bath Asylum, Wells.

1894 Walker, A. Stodart, M.B., 30, Walker Street, Edinburgh.

O.M. Waller, Augustus, M.D., F.R.S., 16, Grove End Road, N.W. (C. 1894-97.)

O.M. Ward, J., D.Sc., Trinity College, Cambridge.

,, Warner, Francis, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1892 Washbourn, J. W., M.D., F.R.C.P., 6, Cavendish Place, W.

O.M. Watteville, A. de, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896; Sec., 1886-9; Editor of *Brain*.)

1897 Weber, F. Parkes, M.D., M.R.C.P., 19, Harley Street, W.

1897 West, Samuel, M.D., F.R.C.P., 43, Wimpole Street, W.

1897 White, Ernest W., M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. White, W. Hale, M.D., F.R.C.P., 65, Harley Street, W. (C. 1897.)

1894 Whiting, Arthur J., M.D., M.R.C.P., 142, Harley Street, W.

1889 Wigglesworth, Joseph, M.D., M.R.C.P., County Asylum, Rainhill, Lancashire.

1894 Williamson, Richard T., M.D., M.R.C.P., 294, Oxford Road, Manchester.

O.M. Wilks, Sir Samuel, Bart., M.D., F.R.S., 72, Grosvenor Street, W. (Pres., 1887; V.P., 1886; C., 1888-91.)

1896 Wilson, Albert, M.D.Edin., M.R.C.P., 33, Fairlop Road, Leytonstone.

,, Wood, Guy M., M.B., M.R.C.P., County Asylum, Rainhill, Lancashire.

1889 Wood, T. Outterson, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 Woodhead, G. Sims, M.D., F.R.C.P.Ed., Examination Hall, Victoria Embankment.

1898 Young, Robert Arthur, M.D., B.Sc., M.R.C.P., 37, Trevor Sq., S.W.

XUM